

Alcohol use and burden for 195 countries and territories, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016

GBD 2016 Alcohol Collaborators*



Summary

Background Alcohol use is a leading risk factor for death and disability, but its overall association with health remains complex given the possible protective effects of moderate alcohol consumption on some conditions. With our comprehensive approach to health accounting within the Global Burden of Diseases, Injuries, and Risk Factors Study 2016, we generated improved estimates of alcohol use and alcohol-attributable deaths and disability-adjusted life-years (DALYs) for 195 locations from 1990 to 2016, for both sexes and for 5-year age groups between the ages of 15 years and 95 years and older.

Methods Using 694 data sources of individual and population-level alcohol consumption, along with 592 prospective and retrospective studies on the risk of alcohol use, we produced estimates of the prevalence of current drinking, abstinence, the distribution of alcohol consumption among current drinkers in standard drinks daily (defined as 10 g of pure ethyl alcohol), and alcohol-attributable deaths and DALYs. We made several methodological improvements compared with previous estimates: first, we adjusted alcohol sales estimates to take into account tourist and unrecorded consumption; second, we did a new meta-analysis of relative risks for 23 health outcomes associated with alcohol use; and third, we developed a new method to quantify the level of alcohol consumption that minimises the overall risk to individual health.

Findings Globally, alcohol use was the seventh leading risk factor for both deaths and DALYs in 2016, accounting for 2.2% (95% uncertainty interval [UI] 1.5–3.0) of age-standardised female deaths and 6.8% (5.8–8.0) of age-standardised male deaths. Among the population aged 15–49 years, alcohol use was the leading risk factor globally in 2016, with 3.8% (95% UI 3.2–4.3) of female deaths and 12.2% (10.8–13.6) of male deaths attributable to alcohol use. For the population aged 15–49 years, female attributable DALYs were 2.3% (95% UI 2.0–2.6) and male attributable DALYs were 8.9% (7.8–9.9). The three leading causes of attributable deaths in this age group were tuberculosis (1.4% [95% UI 1.0–1.7] of total deaths), road injuries (1.2% [0.7–1.9]), and self-harm (1.1% [0.6–1.5]). For populations aged 50 years and older, cancers accounted for a large proportion of total alcohol-attributable deaths in 2016, constituting 27.1% (95% UI 21.2–33.3) of total alcohol-attributable female deaths and 18.9% (15.3–22.6) of male deaths. The level of alcohol consumption that minimised harm across health outcomes was zero (95% UI 0.0–0.8) standard drinks per week.

Interpretation Alcohol use is a leading risk factor for global disease burden and causes substantial health loss. We found that the risk of all-cause mortality, and of cancers specifically, rises with increasing levels of consumption, and the level of consumption that minimises health loss is zero. These results suggest that alcohol control policies might need to be revised worldwide, refocusing on efforts to lower overall population-level consumption.

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Introduction

Alcohol use has a complex association with health. Researchers have recognised alcohol use as a leading risk factor for disease burden, and studies link its consumption to 60 acute and chronic diseases.^{1–3} Additionally, some research suggests that low levels of alcohol consumption can have a protective effect on ischaemic heart disease, diabetes, and several other outcomes.^{4–6} This finding remains an open question, and recent studies have challenged this view by use of mendelian randomisation and meta-analyses.^{7–10}

Determination of harm due to alcohol use is complicated further by the multiple mechanisms through which alcohol use affects health: through cumulative consumption leading to adverse effects on organs and tissues; by acute intoxication leading to injuries or poisoning; and by dependent drinking leading to impairments and potentially self-harm or violence. These effects are also influenced by an individual's consumption volume and pattern of drinking.² Measuring the health effects of alcohol use requires careful consideration of all these factors.

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*Collaborators listed at the end of the Article

Correspondence to:

Prof Emmanuela Gakidou, Institute for Health Metrics and Evaluation, University of Washington, Seattle, WA 98121, USA

gakidou@uw.edu

Research in context

Evidence before this study

Although researchers recognise alcohol use as a leading risk factor for premature death and disability, some evidence suggests that low intake might have a protective effect on specific conditions such as ischaemic heart disease and diabetes. Monitoring of consumption behaviour is required to analyse the health effects of alcohol use. Historically, researchers have relied on self-reported survey data to estimate consumption levels and trends. However, these data have systematic biases that make cross-country comparisons unreliable. The Global Status Report on Alcohol and Health, as well as previous iterations of the Global Burden of Diseases, Injuries, and Risk Factors Study, have sought to produce harmonised, cross-country comparisons of alcohol consumption and its harms, by leveraging data on alcohol sales, the prevalence of current drinking and abstinence, and self-reports of consumption amounts.

Added value of this study

In this analysis we improved available estimates of alcohol use and its associated health burden in five ways. First, we consolidated 694 individual and population-level data sources to estimate alcohol consumption levels among current drinkers. Second, we

developed a method to adjust population-level consumption for alcohol consumed by tourists. Third, we improved pre-existing methods that account for unrecorded population-level consumption. Fourth, we did a new systematic review and meta-analysis of alcohol use and 23 associated health outcomes, which we used to estimate new dose–response curves of relative risk. Fifth, using the new relative risk curves and a new analytical method, we estimated the exposure of alcohol consumption that minimises an individual's total attributable risk.

Implications of all the available evidence

The total attributable burden of alcohol use was larger than previous evidence has indicated and increases monotonically with consumption. Based on weighted relative risk curves for each health outcome associated with alcohol use, the level of consumption that minimises health loss due to alcohol use is zero. These findings strongly suggest that alcohol control policies should aim to reduce total population-level consumption. To potentially reduce the effects of alcohol use on future health loss, there is a need for countries to revisit their alcohol control policies and assess how they can be modified to further lower population-level consumption.

Several studies have attempted to address these factors to provide global estimates of alcohol consumption and its associated health effects. The most comprehensive among these studies have been WHO's Global Status Report on Alcohol and Health, as well as previous iterations of the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD).^{11–13} The present study aims to build upon pre-existing work and to address several limitations found in earlier research.

First, the available studies have assessed the risk of alcohol use by relying on external meta-analyses, which do not control for confounding in the selection of the reference category within constituent studies. This approach is problematic because of the so-called sick quitter hypothesis, which emphasises the importance of reference category selection in correctly assessing risk among drinkers, along with other confounding study characteristics such as survival bias.^{8,14–17} Until recently, most meta-analyses of alcohol consumption have not controlled for the composition of the reference category. Subsequently, assessments of harm relying on these studies have been biased. We sought to resolve this issue within our meta-analyses by including controls for different reference categories and the average age of participants.

Second, previous studies have used sales data to estimate population-level alcohol stock. Researchers have noted the benefit of using sales data instead of survey data for quantifying alcohol stock available within a location.^{18,19} However, sales data still have bias because of consumption by tourists and unrecorded consumption from illicit sales,

home brewing, and local beverages. Without correction for these factors, estimates relying on sales data can be biased and lead to inaccurate cross-national comparisons. In the current study, we adjusted the estimates of population-level alcohol stock to account for the effects of tourism and unrecorded consumption.

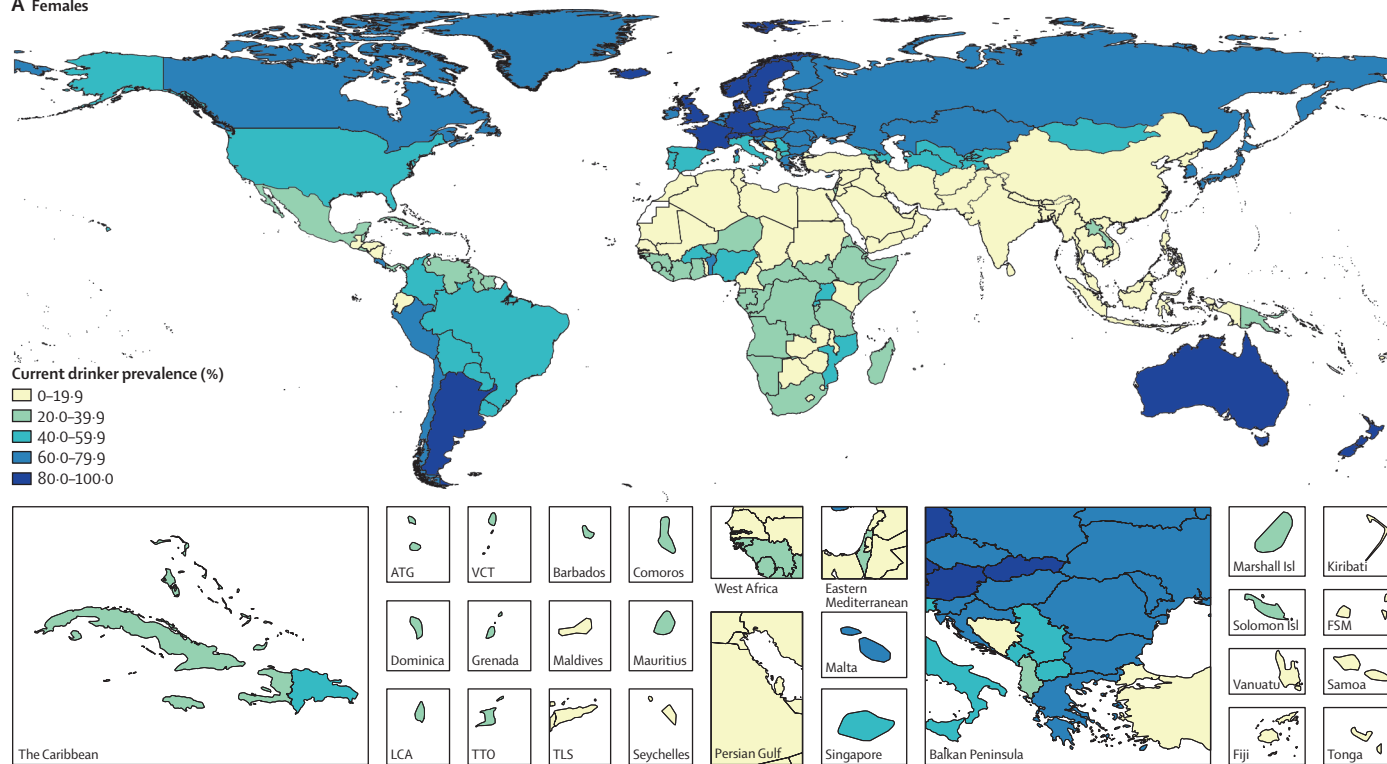
Third, previous studies have assumed zero as the counterfactual exposure level that minimises harm. Within a comparative risk assessment approach, a counterfactual level of consumption that minimises harm is required to estimate population attributable fractions (PAFs).¹ However, this counterfactual level needs to be estimated, rather than assumed, given the complexities involved in estimating the risk of alcohol use across outcomes. Relying on this assumption can fail to capture any potential non-linear effects between alcohol use and health. Our study proposes a new method for the use of available evidence to establish a counterfactual level of exposure across varied relative risks, which provides tangible evidence for low-risk drinking recommendations.

In the present study, we aimed to address these limitations and provide the best available estimates of alcohol use and the associated health burden. We estimated the

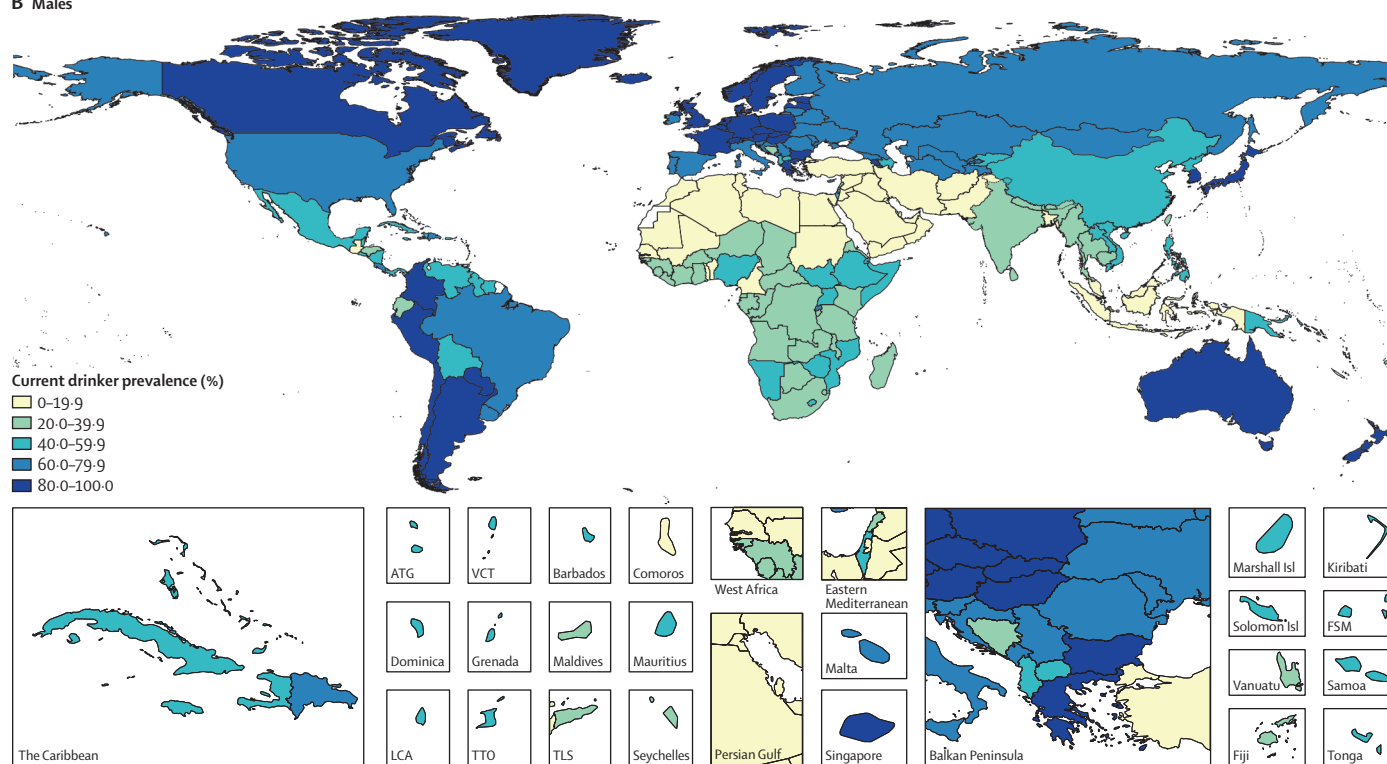
Figure 1: Age-standardised prevalence of current drinking for females (A) and males (B) in 2016, in 195 locations

Current drinkers are defined as individuals who reported having consumed alcohol within the past 12 months. ATG=Antigua and Barbuda. VCT=Saint Vincent and the Grenadines. Isl=Islands. FSM=Federated States of Micronesia. LCA=Saint Lucia. TTO=Trinidad and Tobago. TLS=Timor-Leste.

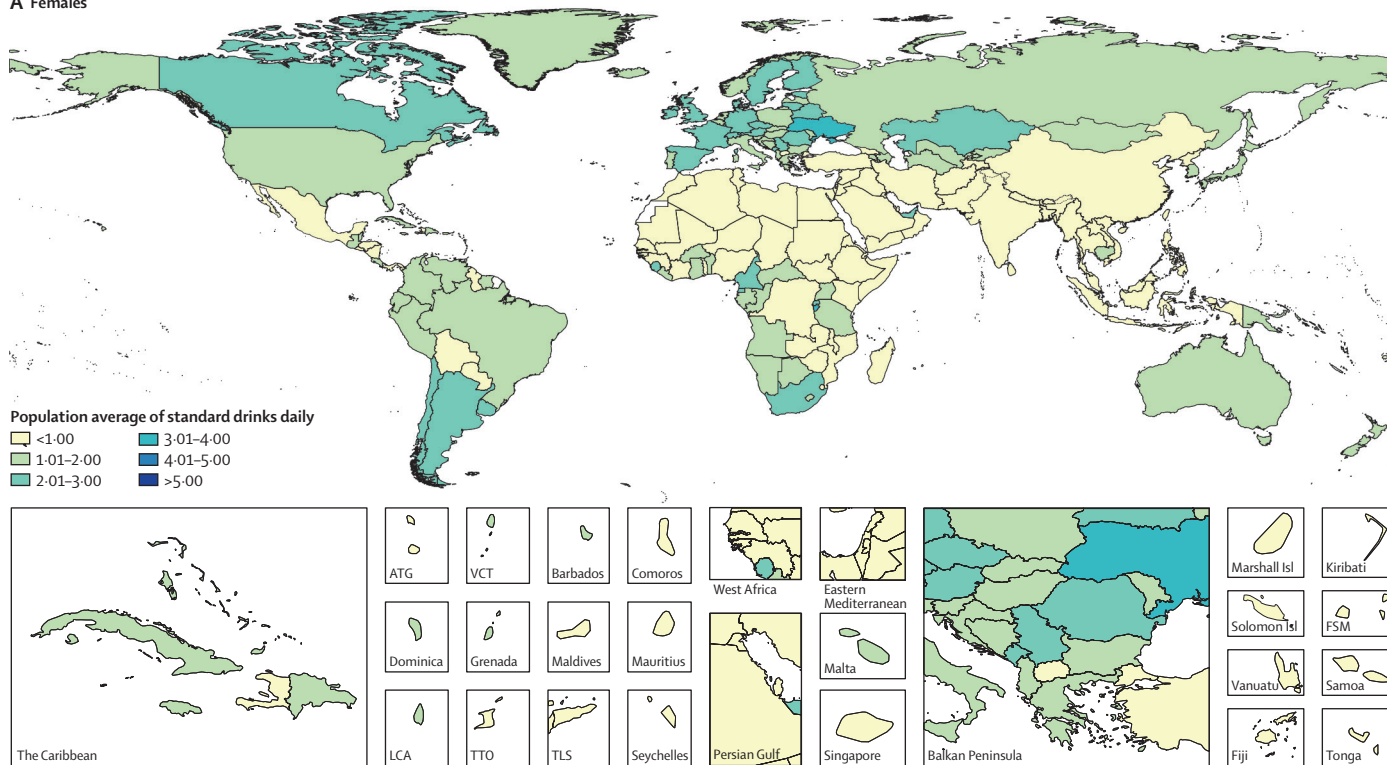
A Females



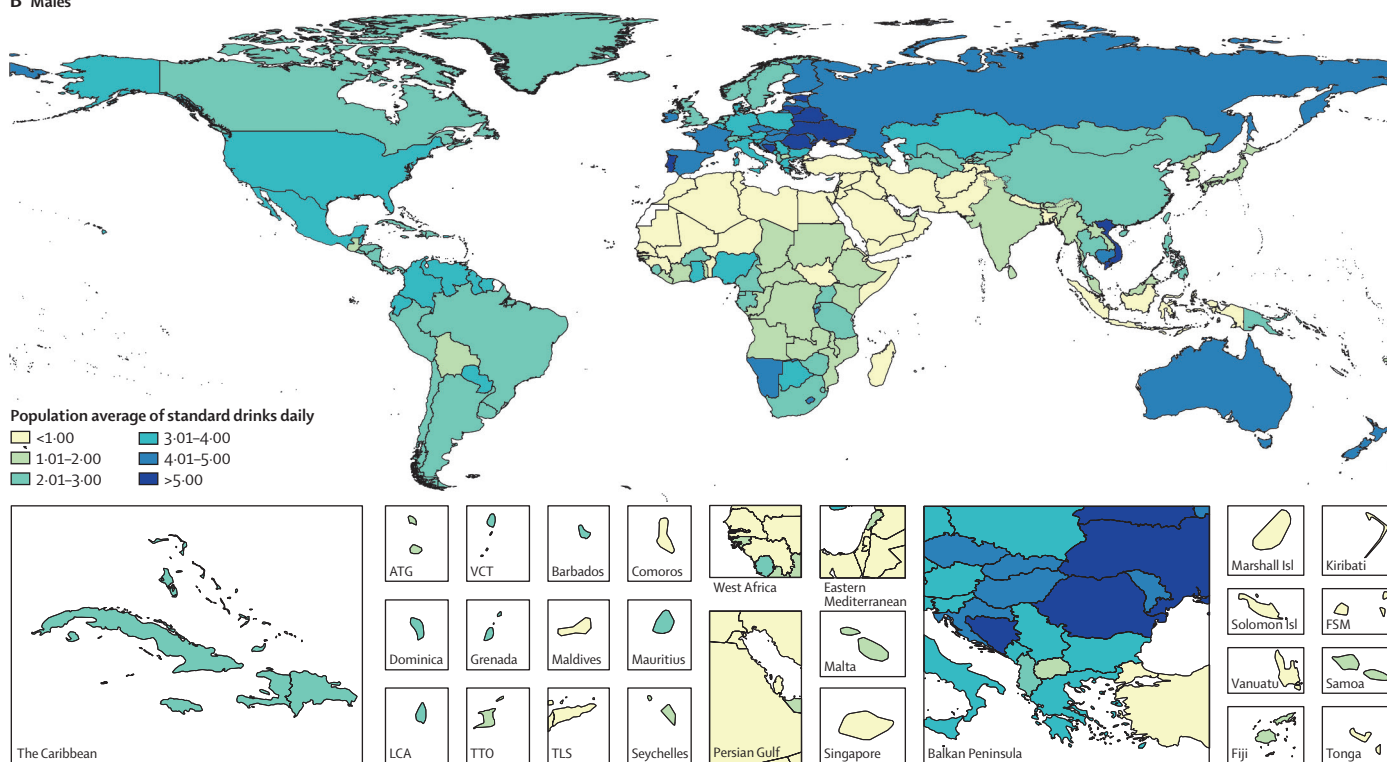
B Males



A Females



B Males



prevalence of current drinking (having one or more drinks in the past year); abstinence from alcohol (having no alcohol in the past year); the distribution of alcohol consumption among current drinkers in standard drinks daily; and the disease burden attributable to alcohol use, in terms of deaths and disability-adjusted life-years (DALYs). We produced these estimates for 195 locations from 1990 to 2016, for both sexes and for 5-year age groups between the ages of 15 years and 95 years and older. We also did a new meta-analysis to assess the dose–response risk of alcohol consumption for 23 outcomes. Lastly, we estimated the level of alcohol consumption that minimises an individual's total attributable risk of any health loss.

Methods

Study design

This study follows the comparative risk assessment framework developed in previous iterations of GBD.²⁰ In the following sections, we summarise our methods and briefly present innovations. A full explanation is available in appendix 1. This study fully adheres to the Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER) statement.²¹

We estimated alcohol use exposure as grams of pure ethanol consumed daily by current drinkers (which we present here in terms of standard drinks daily, defined as 10 g of pure ethyl alcohol). We estimated relative risks by dose in grams of pure ethyl alcohol, for each included risk–outcome pair. We ascertained which cause and injury outcomes to include by reviewing prospective and observational studies of alcohol use, and then assessing the causal association using Bradford-Hill's criteria for causation.²² We included 23 outcomes, and the full list of risk–outcome pairs, as well as the corresponding data sources, are provided in appendix 1 (pp 52–140).

Data sources

We found sources that included indicators of current drinking prevalence and alcohol consumed in grams per day using the Global Health Data Exchange (GHDx) and PubMed.²³ For the meta-analysis, we searched PubMed, the GHDx, and references of previously published meta-analyses. For our exposure estimates, we extracted 121 029 data points from 694 sources across all exposure indicators. For our relative risk estimates, we extracted 3992 relative risk estimates across 592 studies. These relative risk estimates corresponded to a combined study population of 28 million individuals and 649 000 registered

cases of respective outcomes. We list all the included data sources in appendix 1 (pp 52–140).

To estimate standard drinks consumed daily by current drinkers, we followed the general approach used by Rehm and colleagues.¹⁸ We briefly explain this method here, along with two methodological innovations to account for bias in the sales model: an adjustment to account for tourist consumption and an updated adjustment for unrecorded consumption. A full explanation of this approach is available in appendix 1 (pp 18–49).

To estimate exposure, we combined estimates of population-level alcohol stock and individual-level alcohol consumption to produce standard drinks consumed daily among current drinkers and current drinker prevalence, within a specific location, year, age group, and sex. We started by estimating population-level alcohol stock in litres per capita from sales data, individual-level estimates of the prevalence of current drinkers and abstainers from survey data, and individual-level estimates of the amount of alcohol consumed in grams per day from survey data. Then, for a given location and year, we rescaled age-specific and sex-specific estimates of individual-level consumption so that they aggregated to the estimates of population-level consumption. When surveys reported amount consumed in terms of beverage types, we converted these data into grams of pure ethanol using density equations and assumptions of the average alcohol content by drink type (appendix 1, p 50). Finally, we rescaled estimates of current drinking and abstinence so that, within a given location, year, age group, and sex, the two estimates summed to one.

After we derived our model of population-level alcohol stock from sales data, we controlled for sources of bias that could arise from tourism and unrecorded consumption not recorded in formal sales. To account for tourist consumption, we computed an additive measure for alcohol consumed abroad by domestic citizens and subtractive measures for alcohol consumed domestically by tourists. We extracted data on the number of tourists by country of origin and destination from the World Tourism Organization and used these data to obtain estimates of total tourists, percentage of tourists by location, and average duration of stay using a spatio-temporal Gaussian process regression.²⁴ We combined these estimates with measures of alcohol in litres per capita by location, to calculate net amounts of total population-level alcohol stock consumed by tourists or domestic citizens travelling abroad.

To account for alcohol stock not captured within formal alcohol sales data (ie, unrecorded consumption from illicit production, home brewing, local beverages, or alcohol sold as a non-alcohol product), we collated estimates across published studies of the percentage of total alcohol stock due to unrecorded consumption. We sampled 1000 times from a uniform distribution with a range between zero and the average of these collated studies by location (sampling from the uncertainty interval from each study, then averaging the draws) to

See Online for appendix 1

For more on the Global Health Data Exchange see <http://ghdx.healthdata.org/>

Figure 2: Average standard drinks (10 g of pure ethanol per serving) consumed per day, age-standardised, for females (A) and males (B) in 2016, in 195 locations

ATG=Antigua and Barbuda. VCT=Saint Vincent and the Grenadines. Isl=Islands. FSM=Federated States of Micronesia. LCA=Saint Lucia. TTO=Trinidad and Tobago. TLS=Timor-Leste.

generate a conservative estimate of the total stock likely to be unrecorded. We used a conservative approach because of the wide heterogeneity in both the methods and estimates within included data sources. We provide estimates of these percentages in appendix 1 (pp 46–49).

Systematic review and meta-analysis

We did a new systematic review for each associated outcome to incorporate new findings on risk and to improve upon existing approaches. This strategy allowed us to systematically control for reference category confounding in constituent studies across associated outcomes. We provide the search strategy, search diagrams, dose–response curves for each included outcome, and references for each outcome in appendix 1 (pp 57–146).

Drawing from our systematic review, we did a meta-analysis of risk outcomes for alcohol use. For each outcome, we estimated the dose–response relative risk curve using mixed-effects logistic regression with non-linear splines for doses between 0 and 12·5 standard drinks daily. We selected 12·5 standard drinks daily as a cutoff point given the absence of available data beyond this range. We present additional details of the model in appendix 1 (pp 51–138). We tested the significance of including a study-level confounding variable on the composition of the reference category (eg, whether former drinkers were included in the abstainer category or not). When found to be significant, this variable was included as a predictor within the model, which was the case for ischaemic heart disease, ischaemic stroke, and diabetes.

Using our dose–response curves, we estimated the consumption level that minimises harm, which is defined in the comparative risk assessment approach as the theoretical minimum risk exposure level (TMREL). We chose a theoretical minimum on the basis of a weighted average relative risk curve across all attributable outcomes. We constructed weights for each risk outcome based on the respective global, age-standardised DALY rate per 100 000 in 2016 for both sexes. Our TMREL was the minimum of this weighted all-attributable outcome dose–response curve.

Attributable burden due to alcohol use

We calculated PAFs using our estimates of exposure, relative risks, and TMREL, following the same approach taken within the GBD studies.²⁰ For alcohol-use disorders, which are by definition fully attributable, we assumed a PAF of 1.²⁴ Following this calculation, we multiplied PAFs by outcome-specific estimates of deaths and DALYs and summed these across outcomes to calculate the total attributable burden in specific locations. We aggregated both exposure and burden results at the global level and have presented them by quintile of the Socio-demographic Index (SDI). SDI is a summary measure of overall development, based on educational attainment, fertility, and income per capita within a location. Locations categorised by SDI quintile are found in appendix 1

(pp 8–12).²⁵ We also constructed age-standardised values of all estimates, using the same age weights as those used in the GBD standard population.

We made one adjustment to road injury PAFs to estimate how much burden occurred to others because of alcohol use by another individual. We based this adjustment on data from the US Fatality Analysis Reporting System (FARS), which includes the average number of deaths in automobile accidents involving alcohol and the percentage of those deaths distributed by age and sex. We multiplied age-specific and sex-specific alcohol-attributable and road-injury-attributable DALYs by the average number of fatalities, given the driver's age and sex. We then redistributed these attributable DALYs according to the FARS-derived probabilities that a population by age and sex would be involved in a road injury, given the exposed driver's age and sex. Because of data availability, we assumed that locations outside the USA would follow a similar pattern to what we estimated with FARS. After redistributing the attributable DALYs, we derived PAFs again by dividing the redistributed attributable DALYs by total DALYs within specific demographics.

Uncertainty analysis

For all steps, we calculated uncertainty for estimation of exposure, attributable deaths, and DALYs by taking 1000 draws from the data's uncertainty due to sampling error and modelling uncertainty arising from hyperparameter selection and parameter estimation. We then used these draws throughout the entire modelling process. When reporting uncertainty intervals, we present the 2·5th and 97·5th percentiles of the draws.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

Global, regional, and national trends in alcohol consumption

In 2016, 32·5% (95% uncertainty interval [UI] 30·0–35·2) of people globally were current drinkers. 25% (95% UI 23–27) of females were current drinkers, as were 39% (36–43) of males (appendix 2). These percentages corresponded to 2·4 billion (95% UI 2·2–2·6) people globally who were current drinkers, with 1·5 billion (1·4–1·6) male current drinkers and 0·9 billion (0·8–1·0) female current drinkers (appendix 2, pp 2–1994). Globally, the mean amount of alcohol consumed was 0·73 (95% UI 0·68–0·78) standard drinks daily for females and 1·7 (1·5–1·9) standard drinks daily for males.

The prevalence of current drinking varied considerably by location (figure 1). Prevalence was highest for high SDI locations, where 72% (95% UI 69–75) of females and

For more on the US Fatality Analysis Reporting System see <https://www.nhtsa.gov/research-data/fatality-analysis-reporting-system-fars>

See Online for appendix 2

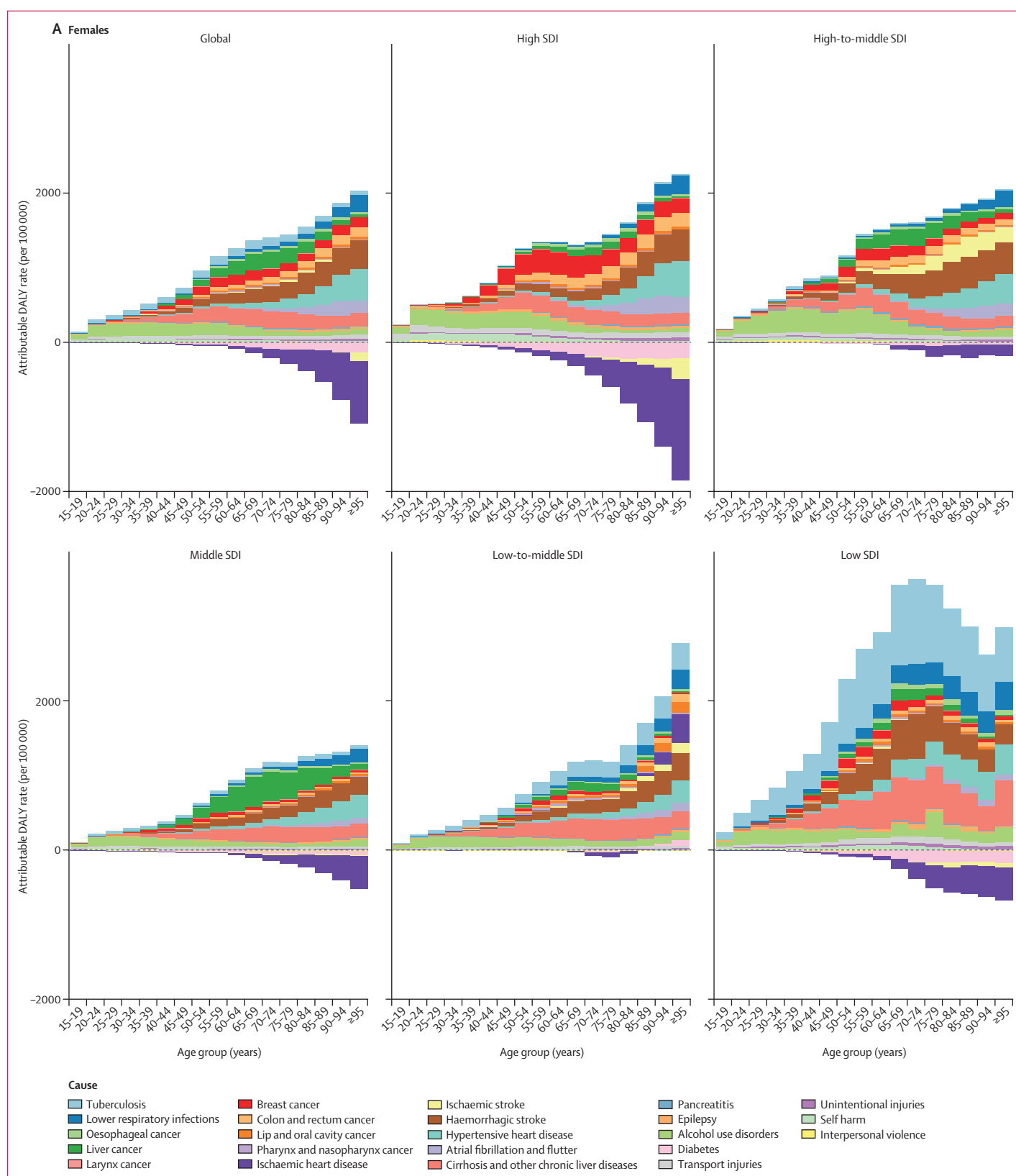
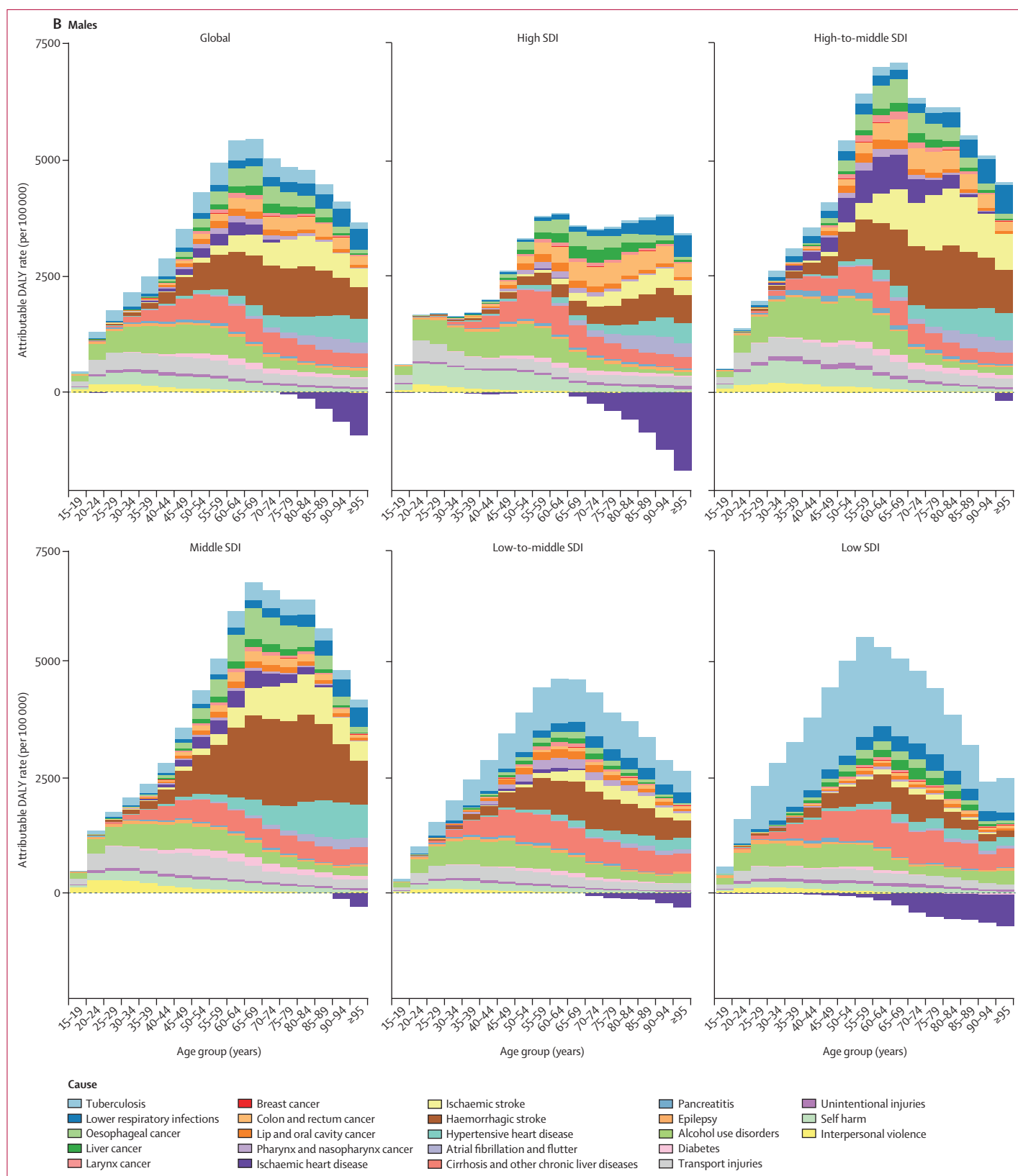


Figure 3 continues on next page



83% (80–85) of males were current drinkers (locations comprising each SDI quintile are provided in appendix 2, pp 8–12). Drinking prevalence was lowest in low-to-middle SDI locations, where 8·9% (95% UI 6·6–9·7) of females and 20% (17–22) of males were current drinkers. Across SDI quintiles, females consumed less alcohol than males, with the size of this disparity decreasing with higher levels of SDI. For example, we found large differences between females and males in Nepal, with only 1·5% (95% UI 1·2–1·9) of females being current drinkers in 2016, compared with 21% (17–25) of males. Conversely, many high SDI locations had similar prevalence between sexes. For example, we found minimal differences in Sweden, where 86% (95% UI 84–88) of females and 87% (85–89) of males were current drinkers.

The population average of standard drinks consumed daily among current drinkers in 2016 also differed widely by location and sex (figure 2). High SDI locations had the highest average of standard drinks consumed daily, with 1·9 (95% UI 1·3–2·7) standard drinks consumed daily among females and 2·9 (2·0–4·1) among males. Low SDI locations had the smallest average for males, with 1·4 (0·6–2·4) standard drinks consumed daily, while low-to-middle SDI locations had the lowest average for females, with 0·3 (0·1–0·6) standard drinks consumed daily.

Global patterns in alcohol-attributable deaths and disease burden

In 2016, 2·8 million deaths (95% UI 2·4–3·3) were attributed to alcohol use. This corresponds to 2·2% (95% UI 1·5–3·0) of total age-standardised deaths among females and 6·8% (5·8–8·0) among males. In terms of overall disease burden, alcohol use led to 1·6% (95% UI 1·4–2·0) of total DALYs globally in 2016 among females and 6·0% (5·4–6·7) among males. Globally, alcohol use was ranked as the seventh leading risk factor for premature death and disability in 2016, compared with other risk factors in the GBD studies. Among the population aged 15–49 years, alcohol use was the leading global risk factor for risk-attributable disease burden, causing 8·9% (95% UI 7·8–9·9) of attributable DALYs for men and 2·3% (2·0–2·6) for women. Among the population aged 15–49 years, 3·8% (95% UI 3·2–4·3) of female deaths and 12·2% (10·8–13·6) of male deaths were attributable to alcohol use.

Both total burden attributable to alcohol use and the proportion of causes associated with alcohol use varied by sex, age, and SDI quintile (figure 3; appendix 2, pp 1997–2186). In absolute terms, the alcohol-attributable

burden by age was smaller for females than for males (figure 3). For females, the alcohol-attributable burden increased with age, while for males the burden increased until between 55–65 years of age, after which attributable burden decreased. Females, particularly in high SDI locations, experienced some protective effects for ischaemic heart disease and diabetes beyond 60 years of age. For males, only high SDI and low SDI locations had noticeable protective effects for ischaemic heart disease, but the effect was small compared with the total attributable burden in those locations.

For both males and females, health outcomes comprising the attributable burden changed across the life-span (figure 3). The three leading causes of attributable deaths in this age group were tuberculosis (1·4% [95% UI 1·0–1·7] of total deaths), road injuries (1·2% [0·7–1·9]), and self-harm (1·1% [0·6–1·5]). For females aged 15–49 years, alcohol use disorders constituted the largest proportion of the attributable burden across SDI quintiles; the primary exception was tuberculosis, which accounted for the largest proportion of attributable burden in low SDI settings. In this age range, transport injuries and alcohol use disorders were the predominant causes of attributable burden for males in high-to-middle SDI quintiles; for low-to-middle SDI and low SDI quintiles, tuberculosis was the primary cause of the attributable burden for both sexes.

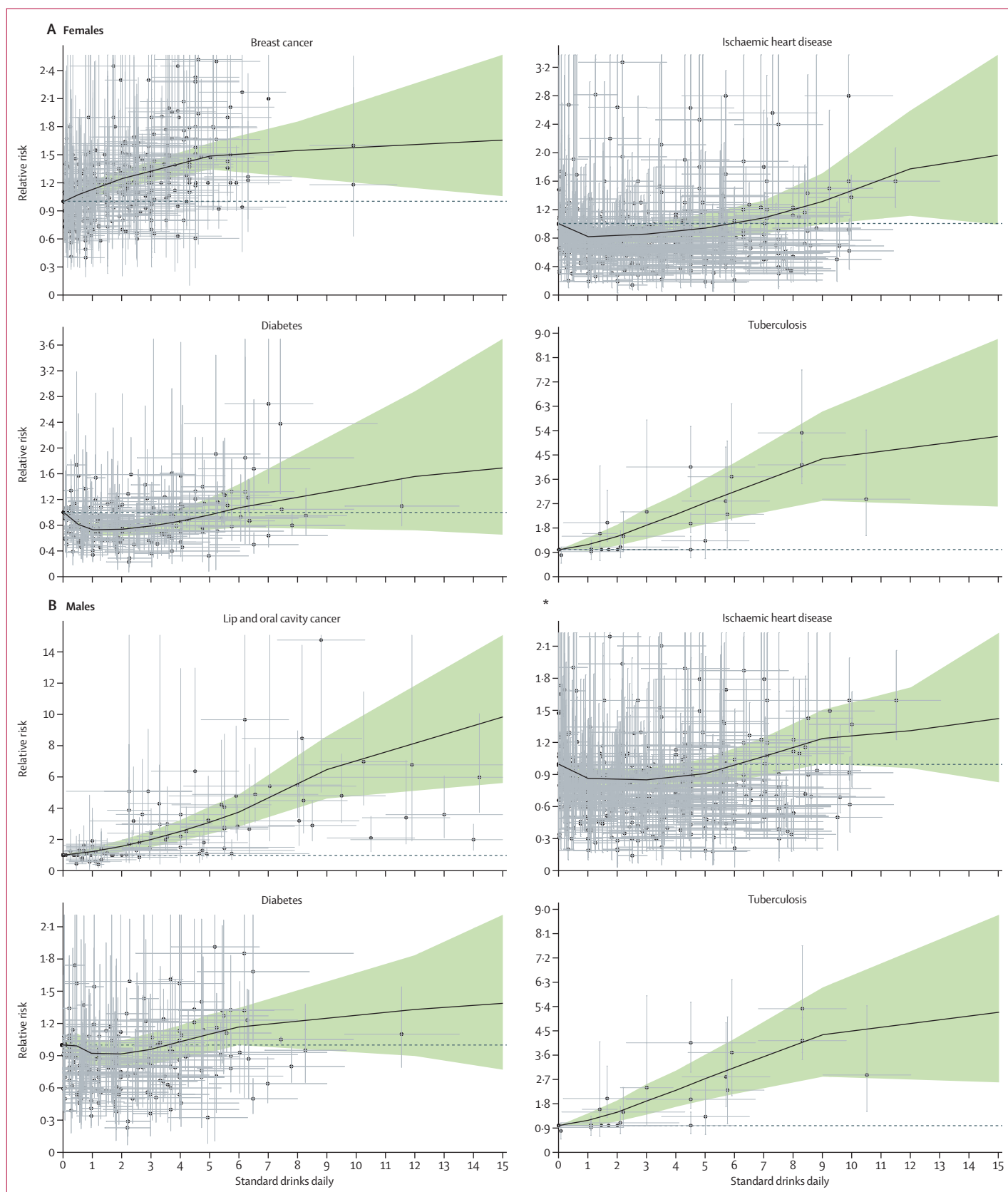
Beyond 50 years of age, the causes of total attributable burden became more complex by SDI quintile. For populations aged 50 years and older, cancers accounted for a large proportion of total alcohol-attributable deaths in 2016, constituting 27·1% (95% UI 21·2–33·3) of total alcohol-attributable female deaths and 18·9% (15·3–22·6) of alcohol-attributable male deaths. In high SDI countries, cancers were the predominant source of attributable burden among both sexes. In low SDI countries, tuberculosis was the primary cause of burden for both sexes, followed by cirrhosis and other chronic liver diseases. The profile of attributable burden in high-to-middle SDI and middle SDI countries for females and males was largely composed of ischaemic stroke and haemorrhagic stroke, followed by liver cancer for females. In all SDI quintiles, haemorrhagic stroke and hypertensive heart disease were the largest sources of burden for females aged 80 years and older. For men in this age group, the composition of the burden was similar to that of males aged 50 years or older.

Health risks associated with alcohol consumption

Figure 4 shows the relative risk curves for selected health outcomes, separately for females and males. Estimated relative risk curves for each health outcome are presented in appendix 2 (pp 52–140). With this analysis, we only found statistically significant evidence for the J-shaped curve for ischaemic heart disease; non-significant J-shaped curves were observed for diabetes and ischaemic stroke. For ischaemic heart disease, we found a

Figure 3: Attributable DALY rate disaggregated by outcome, shown globally and for each region, by age and sex, in 2016

(A) Females. (B) Males. DALY=disability-adjusted life-year. SDI=Socio-demographic Index.



minimum relative risk of 0.86 (0.80–0.96) for men and 0.82 (0.72–0.95) for women, occurring at 0.83 standard drinks daily for men and 0.92 standard drinks daily for women. We found no significant difference in relative risk curves for ischaemic heart disease or diabetes when estimating the curves by age. For all other outcomes, including all cancers, we found that relative risk monotonically increased with alcohol consumption (appendix 2, pp 57–146).

In estimating the weighted relative risk curve, we found that consuming zero (95% UI 0.0–0.8) standard drinks daily minimised the overall risk of all health loss (figure 5). The risk rose monotonically with increasing amounts of daily drinking. This weighted relative risk curve took into account the protective effects of alcohol use associated with ischaemic heart disease and diabetes in females. However, these protective effects were offset by the risks associated with cancers, which increased monotonically with consumption. In a sensitivity analysis, where we explored how the weighted relative risk curve changed on the basis of the choice of weights for various health outcomes, the curve changed significantly only in settings where diabetes and ischaemic heart disease comprised more than 60% of total deaths in a population.

Discussion

In 2016, alcohol use led to 2.8 million deaths and was the leading risk factor for premature death and disability among people aged 15–49 years, with nearly 9% of all attributable DALYs for men and more than 2% for women. Our findings indicate that alcohol use was associated with far more health loss for males than for females, with the attributable burden for men around three times higher than that for women in 2016. By evaluating all associated relative risks for alcohol use, we found that consuming zero standard drinks daily minimises the overall risk to health.

Previous research has analysed all-cause risk due to alcohol use by either investigating all-cause risk in particular cohorts and survey series, or through meta-analyses of those studies.^{26,27} Past findings subsequently suggested a persistent protective effect for some low or moderate levels of alcohol consumption on all-cause

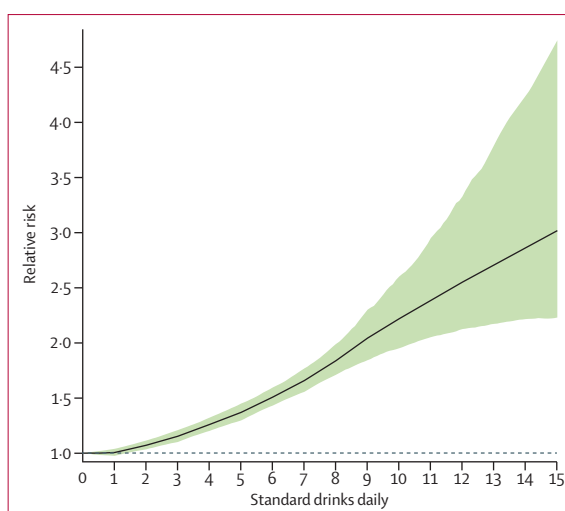


Figure 5: Weighted relative risk of alcohol for all attributable causes, by standard drinks consumed per day
Age-standardised weights determined by the DALY rate in 2016, for both sexes. The dotted line is a reference line for a relative risk of 1. DALY=disability-adjusted life-year.

mortality. However, these studies were limited by small sample sizes, inadequate control for confounders, and non-optimal choices of a reference category for calculating relative risks. More recent research, which has used methodologies such as mendelian randomisation, pooling cohort studies, and multivariable adjusted meta-analyses, increasingly shows either a non-significant or no protective effect of drinking on all-cause mortality or cardiovascular outcomes.^{7,14,28} Our results on the weighted attributable risk are consistent with this body of work. Taken together, these findings emphasise that alcohol use, regardless of amount, leads to health loss across populations. Although we found some protective effects for ischaemic heart disease and diabetes among women, these effects were offset when overall health risks were considered—especially because of the strong association between alcohol consumption and the risk of cancer, injuries, and communicable disease. These findings stress the importance of assessing how alcohol use affects population health across the lifespan.

Evaluating attributable burden across SDI quintiles revealed the magnitude by which outcomes of alcohol use differ and how total attributable burden relates to increasing SDI. Our results indicate that alcohol use and its harmful effects on health could become an increasing challenge amid gains in SDI. Given that most low and low-to-middle SDI settings currently have lower average alcohol consumption than high-to-middle SDI settings, it is crucial for decision makers and government agencies to enact or maintain strong alcohol control policies today to prevent the potential for rising alcohol use in the future. Effective policies now could yield substantial population health benefits for years to come.

Figure 4: Relative risk curves for selected conditions by number of standard drinks consumed daily

(A) Relative risk curves for breast cancer, ischaemic heart disease, diabetes, and tuberculosis for females. (B) Relative risk curves for lip and oral cavity cancer, ischaemic heart disease, diabetes, and tuberculosis for males. Points are relative risk estimates from studies. The vertical and horizontal bars capture the uncertainty in each study, related to the sample size and number of drinks consumed by individuals in the study. The black line represents the estimated relative risk for each condition at each level of consumption. The shaded green areas represent the 95% uncertainty interval associated with the estimated relative risk. The dotted line is a reference line for a relative risk of 1. The relative risk curves for all other health outcomes associated with alcohol use are presented in appendix 2 (pp 57–146).

Our results point to a need to revisit alcohol control policies and health programmes, and to consider recommendations for abstinence. In terms of reducing population-level alcohol use, WHO provides a set of best buys—policies that provide an individual year of healthy life at less than the cost of the average individual income.²⁹ Governments should consider how these recommendations can be implemented within their local contexts and broader policy platforms, including excise taxes on alcohol, controlling the physical availability of alcohol and the hours of sale, and controlling alcohol advertising. Any of these policy actions would contribute to reductions in population-level consumption—an important step toward decreasing the health loss associated with alcohol use.

Failing to address harms from alcohol use, particularly at high levels of consumption, can have dire effects on population health. The mortality crisis in Russia is a striking example, where alcohol use was the primary culprit of increases in mortality starting in the 1980s and led to 75% of deaths among men aged 15–55 years.³⁰ Current global trends—namely, population ageing—portend a growing toll of the alcohol-attributable burden in the absence of policies, particularly since many cancers disproportionately affect older individuals. Consequently, low-to-middle SDI countries could benefit from policy action today to keep alcohol consumption low and prevent greater health loss in the future. High and high-to-middle SDI locations need to consider stronger alcohol reduction policies, such as those recommended by WHO, in an effort to reduce population-level consumption.

Our results should be interpreted within the context of the study's limitations. First, our consumption estimates might not fully capture illicit production or unrecorded consumption given our use of sales data in estimation. We have sought to adjust for consumption beyond sales data, but given the heterogeneity of these estimates it is likely that additional methodological refinements are necessary to improve the quantification of unrecorded consumption. Second, drinking patterns within a year are assumed to be consistent; however, past work shows that drinking patterns, rather than average levels of consumption such as standard daily drinks, might be related to different levels of risk and harm. Unfortunately, the data requirements for assessment of such drinking patterns by age, sex, and location far exceed what is currently available. For instance, few prospective studies quantify the effects of drinking patterns and average levels of consumption in tandem, a requirement for correctly assessing the risk of alcohol-attributable outcomes. Third, the data used to estimate motor vehicle harm caused to others from alcohol use were only available for the USA (ie, FARS data). Although it is unlikely that the patterns observed in FARS are drastically different from those of other locations (appendix 1, pp 141–144), this assumption needs to be tested with

more location-specific estimates. Fourth, we were unable to find robust data about the harm caused to others from alcohol-attributable interpersonal violence, a major potential source of health loss. More retrospective studies are needed to assess the harm to others caused through an individual's alcohol use.³⁰ Fifth, consumption for populations younger than 15 years was not assessed because of data sparseness on alcohol use for these age groups. In the absence of such data, potential approaches to address this limitation, such as assuming consumption patterns of older age groups or trying to extrapolate past levels of alcohol consumption, are likely to introduce additional bias or error. More research on youth drinking and the associated risk is required to estimate alcohol-attributable burden for this age group. Last, we sought to quantify the risk of alcohol use only for outcomes with evidence meeting the criteria for the comparative risk assessment approach of GBD studies. However, there are additional outcomes, such as dementia and psoriasis, for which accumulating evidence suggests that alcohol use might be a risk factor.^{31–33} In combination, these limitations suggest that our results are likely to underestimate both the health risks and overall attributable burden of alcohol use.

Conclusion

Alcohol use is a leading risk factor for disease burden worldwide, accounting for nearly 10% of global deaths among populations aged 15–49 years, and poses dire ramifications for future population health in the absence of policy action today. The widely held view of the health benefits of alcohol needs revising, particularly as improved methods and analyses continue to show how much alcohol use contributes to global death and disability. Our results show that the safest level of drinking is none. This level is in conflict with most health guidelines, which espouse health benefits associated with consuming up to two drinks per day. Alcohol use contributes to health loss from many causes and exacts its toll across the lifespan, particularly among men. Policies that focus on reducing population-level consumption will be most effective in reducing the health loss from alcohol use.

GBD 2016 Alcohol Collaborators

Max G Griswold, Nancy Fullman, Caitlin Hawley, Nicholas Arian, Stephanie R M Zimsen, Hayley D Tymeson, Vidhya Venkateswaran, Austin Douglas Tapp, Mohammad H Forouzanfar, Joseph S Salama, Kalkidan Hassen Abate, Degu Abate, Solomon M Abay, Cristiana Abbafati, Rizwan Suliankatchi Abdulkader, Zegeye Abebe, Victor Aboyans, Mohammed Mehdi Abrar, Pawan Acharya, Olatunji O Adetokunboh, Tara Ballav Adhikari, Jose C Adsuar, Mohsen Afarideh, Emilie Elisabet Agardh, Gina Agarwal, Sargis Aghasi Aghayan, Sutapa Agrawal, Muktar Beshir Ahmed, Mohammed Akibu, Tomi Akinjemiju, Nadia Akseer, Deena H Al Asfour, Ziyad Al-Aly, Fares Alahdab, Khurshid Alam, Ammar Albujee, Kefyalew Addis Alene, Raghib Ali, Syed Danish Ali, Mehran Alijanzadeh, Syed Mohamed Aljunid, Ala'a Alkerwi, Peter Allebeck, Nelson Alvis-Guzman, Azmeraw T Amare, Leopold N Aminde, Walid Ammar, Yaw Ampem Amoako, Gianna Gayle Herrera Amul, Catalina Liliana Andrei, Colin Angus, Mustafa Geleto Ansha,

Carl Abelardo T Antonio, Olatunde Aremu, Johan Årnlöv, Al Artaman, Krishna K Aryal, Reza Assadi, Marcel Ausloos, Leticia Avila-Burgos, Euripide F G A Avokpaho, Ashish Awasthi, Henok Tadesse Ayele, Rakesh Ayer, Tambe B Ayuk, Peter S Azzopardi, Hamid Badali, Alaa Badawi, Maciej Banach, Suzanne Lyn Barker-Collo, Lope H Barrero, Huda Basaleem, Estifanos Baye, Shahrzad Bazargan-Hejazi, Neeraj Bedi, Yannick Béjot, Abate Bekele Belachew, Saba Abraham Belay, Derrick A Bennett, Isabela M Bensoron, Eduardo Bernabe, Robert S Bernstein, Addisu Shunu Beyene, Tina Beyranvand, Soumyadeep Bhaumik, Zulfiqar A Bhutta, Belete Biadgo, Ali Bijani, Nigus Bililign, Sait Montes Birlík, Charles Birungi, Hailemichael Bizuneh, Peter Bjerregaard, Tone Bjørge, Guilherme Borges, Cristina Bosetti, Soufiane Boufous, Nicola Luigi Bragazzi, Hermann Brenner, Zahid A Butt, Lucero Cahuana-Hurtado, Bianca Calabria, Ismael R Campos-Nonato, Julio Cesar Campuzano Rincon, Giulia Carreras, Juan J Carrero, Félix Carvalho, Carlos A Castañeda-Orjuela, Jacqueline Castillo Rivas, Ferrán Catalá-López, Jung-Chen Chang, Fiona J Charlson, Aparajita Chattopadhyay, Pankaj Chaturvedi, Rajiv Chowdhury, Devasahayam J Christopher, Sheng-Chia Chung, Liliana G Ciobanu, Rafael M Claro, Sara Conti, Ewerton Cousin, Michael H Criqui, Berihun Assefa Dachew, Paul I Dargan, Ahmad Daryani, José Das Neves, Kairat Davletov, Filipa De Castro, Barbora De Courten, Jan-Walter De Neve, Louisa Degenhardt, Gebre Teklemariam Demoz, Don C Des Jarlais, Subhojit Dey, Rupinder Singh Dhaliwal, Samath Dhamminda Dharmaratne, Meghnath Dhimai, David Teye Doku, Kerrie E Doyle, Manisha Dubey, Eleonora Dubljanin, Bruce B Duncan, Hedyeh Ebrahimi, Dumessa Edessa, Maysaa El Sayed Zaki, Sergei Petrovich Ermakov, Holly E Erskine, Alireza Esteghamati, Mahbobeh Faramarzi, Andrea Farioli, Andre Faro, Maryam S Farvid, Farshad Farzadfar, Valery L Feigin, Mariana Santos Felisbino-Mendes, Eduarda Fernandes, Alize J Ferrari, Cleusa P Ferri, Daniel Obadare Fijabi, Irina Filip, Jonas David Finger, Florian Fischer, Abraham D Flaxman, Richard Charles Franklin, Neal D Futran, Silvano Gallus, Morsaleh Ganji, Fortune Gbetoho Gankpe, Gebremedhin Berhe Gebregergs, Tsegaye Tewelde Gebrehiwot, Johanna M Geleijnse, Reza Ghadimi, Lilian A Ghandour, Mamata Ghimire, Paramjit Singh Gill, Ibrahim Abdelmageed Ginawi, Ababi Zergaw Z Giref, Philimon N Gona, Sameer Vali Gopalani, Carolyn C Gotay, Alessandra C Goulart, Felix Greaves, Giuseppe Grosso, Yuming Guo, Rahul Gupta, Rajeev Gupta, Vipin Gupta, Reyna Alma Gutiérrez, Murthy Gvs, Nima Hafezi-Nejad, Tekleberhan Beyene Hagos, Gessesew Bugssa Hailu, Randah R Hamadeh, Samer Hamidi, Graeme J Hankey, Hilda L Harb, Sivadasanpillai Harikrishnan, Josep Maria Haro, Hamid Yimam Hassen, Rasmus Havmoeller, Simon I Hay, Behzad Heibati, Andualem Henok, Ileana Heredia-Pi, Norberto Francisco Hernández-Llanes, Claudiu Herteliu, Desalegn Ts Tsegaw Hibstu, Praveen Hoogar, Nobuyuki Horita, H Dean Hosgood, Mostafa Hosseini, Mihaela Hostiuc, Guoqing Hu, Hsiang Huang, Abdullatif Husseini, Bulat Idrisov, Bogdan Vasile Ileanu, Olayinka Stephen Ilesanmi, Seyed Sina Naghibi Irvani, Sheikh Mohammed Shariful Islam, Maria D Jackson, Mihajlo Jakovljevic, Moti Tolera Jalu, Achala Upendra Jayatilleke, Ravi Prakash Jha, Jost B Jonas, Jacek Jerzy Jozwiak, Zubair Kabir, Rajendra Kadel, Amaha Kahsay, Umesh Kapil, Amir Kasaeian, Tesfaye D Dessale Kassa, Srinivasa Vittal Katikireddi, Norito Kawakami, Seifu Kebede, Adane Teshome Kefale, Peter Njenga Keiyoro, Andre Pascal Kengne, Yousef Khader, Morteza Abdullatif Khafaie, Ibrahim A Khalil, Md Nuruzzaman Khan, Young-Ho Khang, Mona M Khater, Jagdish Khubchandani, Cho-Il Kim, Daniel Kim, Yun Jin Kim, Ruth W Kimokoti, Adnan Kisa, Mika Kivimäki, Sonali Kochhar, Soewarta Kosen, Parvaiz A Koul, Ai Koyanagi, Kewal Krishan, Barthelémy Kuate Defo, Burcu Kucuk Bicer, Veena S Kulkarni, Pushpendra Kumar, Alessandra Lafrancconi, Arjun Lakshmana Balaji, Ratilal Lalloo, Tea Lallukka, Hilton Lam, Faris Hasan Lami, Qing Lan, Justin J Lang, Sonia Lansky, Anders O Larsson, Arman Latifi, Janet L Leasher, Paul H Lee, James Leigh, Mall Leinsalu, Janni Leung, Miriam Levi, Yichong Li, Lee-Ling Lim, Shai Linn, Shiwei Liu, Andrea Lobato-Cordero, Alan D Lopez, Paulo A Lotufo, Erlын Rachelle King Macarayan, Isis Eloah Machado, Fabiana Madotto, Hassan Magdy Abd El Razek, Muhammed Magdy Abd El Razek, Marek Majdan, Reza Majdzadeh, Azeem Majeed, Reza Malekzadeh, Deborah Carvalho Malta, Chabila Christopher Mapoma, Jose Martinez-Raga, Pallab K Maulik, Mohsen Mazidi, Martin Mckee, Varshil Mehta, Toni Meier, Tesfa Mekonen, Kidanu Gebremariam Meles, Addisu Melese, Peter T N Memiah, Walter Mendoza, Desalegn Tadesse Mengistu, George A Mensah, Tuomo J Meretoja, Haftay Berhane Mezgebe, Tomasz Miazgowski, Ted R Miller, Gk Mini, Andreea Mirica, Erkin M Mirrakhimov, Babak Moazen, Karzan Abdulmuhsin Mohammad, Noushin Mohammadifard, Shafiu Mohammed, Lorenzo Monasta, Paula Moraga, Lidia Morawska, Seyyed Meysam Mousavi, Satinath Mukhopadhyay, Kamarul Imran Musa, Aliya Naheed, Gurudatta Naik, Farid Najafi, Vinay Nangia, Jobert Richie Nansseu, Mudavath Siva durga prasad Nayak, Chakib Nejari, Subas Neupane, Sudan Prasad Neupane, Josephine W Ngunjiri, Cuong Tat Nguyen, Long Hoang Nguyen, Trang Huyen Nguyen, Dina Nur Anggraini Ningrum, Yirga Legesse Nirayo, Jean Jacques Noubiap, Richard Ofori-Asenso, Felix Akpojene Ogbo, In-Hwan Oh, Olanrewaju Oladimeji, Andrew T Olagunju, Pedro R Olivares, Bolajoko Olubukunola Olusanya, Jacob Olusegun Olusanya, Anu Mary Oommen, Eyal Oren, Heather M Orpana, Doris D V Ortega-Altamirano, Justin R Ortiz, Erika Ota, Mayowa Ojo Owolabi, Abayomi Samuel Oyekale, Mahesh P A, Adrian Pana, Eun-Kee Park, Charles D H Parry, Hadi Parsian, Ajay Patle, George C Patton, Deepak Paudel, Max Petzold, Michael R Phillips, Julian David Pillay, Maarten J Postma, Farshad Pourmalek, Dorairaj Prabhakaran, Mostafa Qorbani, Amir Radfar, Anwar Rafay, Alireza Rafiei, Fakher Rahim, Afarin Rahimi-Movaghgar, Mahfuzar Rahman, Muhammad Aziz Rahman, Rajesh Kumar Rai, Sasa Rajsic, Sree Bhushan Raju, Usha Ram, Saleem M Rana, Chhabhi Lal Ranabhat, David Laith Rawaf, Salman Rawaf, Robert C Reiner, Cesar Reis, Andre M N Renzaho, Mohammad Sadegh Rezai, Leonardo Roeveer, Luca Ronfani, Robin Room, Gholamreza Roshandel, Ali Rostami, Gregory A Roth, Ambuj Roy, Yogesh Damodar Sabde, Basema Saddik, Saeid Safiri, Amirhossein Sahebkar, Joseph S Salama, Zikria Saleem, Joshua A Salomon, Sundeep Santosh Salvi, Juan Sanabria, Maria Dolores Sanchez-Niño, Damian Francesco Santomauro, Itamar S Santos, Milena M M Santric Milicevic, Abdur Razaque Sarker, Rodrigo Sarmiento-Suárez, Nizal Sarrafzadegan, Benn Sartorius, Maheswar Satpathy, Monika Sawhney, Sonia Saxena, Mete Saylan, Michael P Schaub, Maria Inês Schmidt, Ione J C Schneider, Ben Schöttker, Aletta Elisabeth Schutte, Falk Schwendicke, Sadaf G Sepanlou, Masood Ali Shaikh, Mehdi Sharif, Jun She, Aziz Sheikh, Jiabin Shen, Mekonnen Sisay Shiferaw, Mika Shigematsu, Rahman Shiri, Kawkab Shishani, Ivy Shiue, Sharvari Rahul Shukla, Itamar Dora Sigfusdottir, Diego Augusto Santos Silva, Natacha Torres Da Silva, Dayane Gabriele Alves Silveira, Dharendra Narain Sinha, Freddy Sitas, Adauto Martins Soares Filho, Moslem Soofi, Reed J D Sorensen, Joan B Soriano, Chandrashekhar T Sreeramareddy, Nadine Steckling, Dan J Stein, Mu'awiyah Babale Sufiyan, Patrick J Sur, Bryan L Sykes, Rafael Tabarés-Seisdedos, Takahiro Tabuchi, Mohammad Tavakkoli, Arash Tehrani-Banihashemi, Merhawi Gebremedhin Tekle, Subash Thapa, Nihal Thomas, Roman Topor-Madry, Fotis Topouzis, Bach Xuan Tran, Christopher E Troeger, Thomas Clement Truelsen, Nikolaos Tsilimparis, Stefanos Tyrovolas, Kingsley Nnanna Ukwaja, Irfan Ullah, Olalekan A Uthman, Pascual R Valdez, Job F M Van Boven, Tommi Juhani Vasankari, Narayanaswamy Venketasubramanian, Francesco S Violante, Sergey Konstantinovich Vladimirov, Vasily Vlassov, Stein Emil Vollset, Theo Vos, Fasil Wagnew Shiferaw Wagnew, Yasir Waheed, Yuan-Pang Wang, Elisabete Weiderpass, Fitsum Weldegebreal, Kidu Gidey Weldegewergs, Andrea Werdecker, Ronny Westerman, Harvey A Whiteford, Justyna Widecka, Tissa Wijeratne, Grant M A Wyper, Gelin Xu, Tomohide Yamada, Yuichiro Yano, Pengpeng Ye, Ebrahim M Yimer, Paul Yip, Biruck Desalegn Yirsaw, Engida Yisma, Naohiro Yonemoto, Seok-Jun Yoon, Marcel Yotebieng, Mostafa Z Younis, Geevar Zachariah, Zoubida Zaidi, Mohammad Zamani, Xueying Zhang, Sanjay Zodepy, Ali H Mokdad, Mohsen Naghavi,

Christopher J L Murray, Emmanuela Gakidou.

Affiliations

Institute for Health Metrics and Evaluation (M G Griswold MA, N Fullman MPH, C Hawley MSPH, N Arian BS, S R M Zimsen MA, H D Tymeson BA, A D Tapp BS, J S Salama MS, Prof L Degenhardt PhD, S D Dharmaratne MD, Prof V L Feigin PhD, Prof A D Flaxman PhD, Prof S I Hay DSc, Prof M Jakovljevic PhD, I A Khalil MD, R C Reiner PhD, G A Roth MD, P J Sur BA, C E Troeger MPH, Prof S Vollset DrPH, Prof T Vos PhD, Prof H A Whiteford PhD, Prof A H Mokdad PhD, Prof M Naghavi PhD, Prof C J L Murray DPhil, Prof E Gakidou PhD), Department of Global Health (F J Charlson PhD, S Kochhar MD, Prof J R Ortiz MD, R J D Sorensen MPH), School of Medicine Department of Otolaryngology-Head and Neck Surgery (N D Futran MD), University of Washington, Seattle, WA, USA (Prof E Oren PhD); Department of Epidemiology (V Venkateswaran BDS), Department of Nutrition (M S Farvid PhD), Ariadne Labs (E K Macarayan PhD), BWH Division of General Internal Medicine and Primary Care (Prof A Sheikh MSc), Harvard University, Boston, MA, USA; Seattle Genetics, Seattle, WA, USA (M H Forouzanfar PhD); Department of Population and Family Health (Prof K H Abate PhD), Department of Epidemiology (M B Ahmed MPH, Prof T T Gebrehiwot MPH), Jimma University, Jimma, Ethiopia; School of Public Health (A S Beyene MPH, M G Tekle MPH), School of Pharmacy (D Edessa MSc, Prof M S Shiferaw MSc), Department of Medical Laboratory Science (Prof F Weldegebreel MSc), Haramaya University, Harar, Ethiopia (D Abate MSc, M T Jalu MPH); Department of Pharmacology and Clinical Pharmacy (S M Abay PhD), Department of Reproductive Health and Health Service Management (A Z Z Giref PhD), School of Alaide Health Sciences (Prof E Yisma MPH), Addis Ababa University, Addis Ababa, Ethiopia (M M Abrar MS, G T Demoz MSc); Department of Law Philosophy and Economic Studies, La Sapienza University, Rome, Italy (Prof C Abbafati PhD); Department of Public Health (R S Abdulkader MD), Office of the Undersecretary of Health Affairs (D H Al Asfoor MSc), Ministry of Health, Riyadh, Saudi Arabia; Human Nutrition (D Abebe MSc), Institute of Public Health (Prof K A Alene MPH, Prof B A Dachew MPH), Department of Clinical Chemistry (B Biadgo BSc), University of Gondar, Gondar, Ethiopia; Department of Cardiology, Dupuytren University Hospital, Limoges, France (Prof V Aboyans MD); Institute of Epidemiology, University of Limoges, Limoges, France (Prof V Aboyans MD); Nepal Development Society, Nepal (P Acharya MPH); Department of Global Health (O O Adetokunboh MSc), Department of Psychiatry (Prof C D H Parry PhD), Stellenbosch University, Cape Town, South Africa; Cochrane South Africa (O O Adetokunboh MSc), Unit for Hypertension and Cardiovascular Disease (Prof A E Schutte PhD), South African Medical Research Council, Cape Town, South Africa (Prof D J Stein MD); Nepal Health Research Environment, Center for Social Science and Public Health Research Nepal, Nepal (T B Adhikari MPH); Unit for Health Promotion Research (T B Adhikari MPH), National Institute of Public Health (Prof P Bjerregaard DrPH), Department of Public Health (S Thapa PhD), University of Southern Denmark, Odense, Denmark; Faculty of Sport Science, University of Extremadura, Spain (J C Adsuar PhD); Endocrinology and Metabolism Research Center (M Afarideh MD, H Ebrahimi MD, Prof A Esteghamati MD, M Ganji MD), Digestive Disease Research Institute (H Ebrahimi MD), Non-Communicable Diseases Research Center (F Farzadfar MD, S N Irvani MD), School of Medicine (N Hafezi-Nejad MD), Hematologic Malignancies Research Center (A Kasaeian PhD), Hematology-Oncology and Stem Cell Transplantation Research Center (A Kasaeian PhD), Knowledge Utilization Research Center (KURC) (Prof R Majdzadeh PhD), Digestive Diseases Research Institute (Prof R Malekzadeh MD, G Roshandel PhD, S G Sepanlou MD), Department of Health Management and Economics, School of Public Health (S Mousavi PhD), Iranian National Center for Addiction Studies (INCAS) (Prof A Rahimi-Movaghar MD), Community-Based Participatory-Research Center (CBPR) (Prof R Majdzadeh PhD), Tehran University of Medical Sciences, Tehran, Iran (Prof M Hosseini PhD, Prof F Rahim PhD); Department of Public Health Sciences (Prof E E Agardh PhD, Prof P Allebeck MD), Department of Neurobiology (Prof J Årnlöv PhD), Department of

Medical Epidemiology and Biostatistics (Prof J J Carrero PhD, Prof E Weiderpass PhD), Karolinska Institute, Stockholm, Sweden; Department of Family Medicine, McMaster University, Hamilton, ON, Canada (G Agarwal MD); Chair of Zoology, Yerevan State University, Yerevan, Armenia (S A Aghayan PhD); Research Group of Molecular Parasitology, Scientific Center of Zoology and Hydroecology, Yerevan, Armenia (S A Aghayan PhD); Indian Institute of Public Health (Prof S Zodpey PhD), Public Health Foundation of India (PHFI), Gurugram, India (S Agrawal PhD, A Awasthi PhD, Prof D Prabhakaran DM); Vital Strategies, Gurugram, India (S Agrawal PhD); Department of Midwifery (M Akibu MSc), Department of Public Health, Lexington, KY, USA (M G Ansha MPH); Debre Berhan University, Debre Berhan, Ethiopia; Department of Epidemiology, University of Kentucky, USA (T Akinyemiju PhD); Nutritional Sciences, Faculty of Medicine (A Badawi PhD), Centre for Global Child Health, The Hospital for Sick Children, University of Toronto, Toronto, ON, Canada (N Akseer PhD, Prof Z A Bhutta PhD); Nuffield Department of Population Health, University of Oxford, Oxford, UK (D H Al Asfoor MSc, R Ali MPH, D A Bennett PhD); Department of Internal Medicine, Washington University in St. Louis, Saint Louis, MO, USA (Z Al-Aly MD); VA Saint Louis Health Care System, Clinical Epidemiology Center, Department of Veterans Affairs, Saint Louis, MO, USA (Z Al-Aly MD); Evidence Based Practice Center, Mayo Clinic Foundation for Medical Education and Research, Rochester, MN, USA (Prof F Alahdab MD); Research Committee, Education Committee, and Avicenna Journal of Medicine Editor, Syrian American Medical Society, Washington, Washington DC, USA (Prof F Alahdab MD); School of Population and Global Health (K Alam PhD), Medical School (Prof G J Hankey MD), University of Western Australia, Perth, WA, Australia; Nab'a Al-Hayat Foundation for Medical Sciences and Health Care, Iraq (A Albujeer DDS); Research School of Population Health (Prof K A Alene MPH), National Centre for Epidemiology and Population Health (B Calabria PhD), Australian National University, Canberra, ACT, Australia; Public Health Research Center, New York University Abu Dhabi, Abu Dhabi, United Arab Emirates (R Ali MPH); University of London, Pakistan (S Ali BA); Social Determinants of Health Research Center, Qazvin University of Medical Sciences, Qazvin, Iran (M Alijanzadeh PhD); Department of Health Policy and Management, Kuwait University, Safat, Kuwait (Prof S M Aljunied PhD); International Centre for Casemix and Clinical Coding, National University of Malaysia, Bandar Tun Razak, Malaysia (Prof S M Aljunied PhD); Department of Population Health, Luxembourg Institute of Health, Strassen, Luxembourg (A Alkerwi PhD); Swedish Research Council for Health, Working Life, and Welfare, Stockholm, Sweden (Prof P Allebeck MD); Research Group in Health Economics, University of Cartagena, Cartagena, Colombia (Prof N Alvis-Guzman PhD); Research Group in Hospital Management and Health Policies, University of the Coast, Barranquilla, Colombia (Prof N Alvis-Guzman PhD); Sansom Institute, University of South Australia (A Amare PhD); Wardliparingga Aboriginal Research Unit (P S Azzopardi PhD), South Australian Health and Medical Research Institute, Adelaide, SA, Australia; Psychiatry (Prof T Mekonen MSc), Bahir Dar University, Bahir Dar, Ethiopia (A Amare PhD); School of Public Health (L N Aminde MD, F J Charlson PhD, Prof B A Dachew MPH, H E Erskine PhD, A J Ferrari PhD, D F Santomauro PhD), School of Dentistry (Prof R Lalloo PhD), University of Queensland, Brisbane, QLD, Australia (J Leung PhD, Prof H A Whiteford PhD); Department of the Health Industrial Complex and Innovation in Health (Prof D A Silveira MSc), Department of Diseases and Non-Communicable Diseases and Health Promotion (A M Soares Filho DSc), Federal Ministry of Health, Brasilia, Brazil; Federal Ministry of Health, Beirut, Lebanon (Prof W Ammar PhD); Faculty of Health Sciences (Prof W Ammar PhD), Department of Epidemiology and Population Health (Prof L A Ghandour PhD), American University of Beirut, Beirut, Lebanon; Department of Internal Medicine, Komfo Anokye Teaching Hospital, Kumasi, Ghana (Y A Amoako MD); Lee Kuan Yew School of Public Policy (G G H Amul MSc), Yong Loo Lin School of Medicine (Prof N Venketasubramanian FRCP), National University of Singapore, Singapore, Singapore; Department of Legal Medicine and Bioethics (Prof M Hostiuic PhD), Carol Davila University of Medicine and

Pharmacy, Bucharest, Romania (C Andrei PhD); School of Health and Related Research, University of Sheffield, Sheffield, UK (C Angus MSc); Department of Health Policy and Administration (C T Antonio MD), Development and Communication Studies (E K Macarayan PhD), University of the Philippines Manila, Manila, Philippines; School of Health Sciences, Birmingham City University, Birmingham, England (O Aremu PhD); School of Health and Social Studies, Dalarna University, Falun, Sweden (Prof J Årnlöv PhD); Department of Community Health Sciences, University of Manitoba, Winnipeg, MB, Canada (A Artaman PhD); DFID Nepal Health Sector Programme 3, Monitoring Evaluation and Operational Research Project, ABT Associates Nepal, Lalitpur, Nepal (K K Aryal PhD); Education Development Center (Prof R Assadi PhD), Department of Medical Biotechnology (Prof A Sahebkar PhD), Mashhad University of Medical Sciences, Mashhad, Iran; School of Business, University of Leicester, Leicester, UK (Prof M Ausloos PhD); Center for Health Systems Research (L Avila-Burgos PhD, L Cahuana-Hurtado PhD, Prof I Heredia-Pi PhD, D D V Ortega-Altamirano DrPH), National Institute of Public Health, Cuernavaca, Mexico (I R Campos-Nonato PhD, J Campuzano Rincon PhD, F De Castro PhD); Deworm3 Project, Bénin Clinical Research Institute (IRCB), Calavi, Benin (E F Avokpaho MD); Control of Infectious Diseases Project (E F Avokpaho MD), Non-Communicable Disease Department (F G Gankpe MD), Laboratory of Studies and Research-Action in Health (LERAS), Porto-Novo, Benin; Indian Institute of Public Health, Gandhinagar, India (A Awasthi PhD); Department of Epidemiology, Biostatistics, and Occupational Health, McGill University, Montreal, QC, Canada (H T Ayele PhD); Department of Public Health, Dilla University, Dilla, Ethiopia (H T Ayele PhD); Department of Community and Global Health (R Ayer MSc), Department of Mental Health (Prof N Kawakami PhD), Department of Diabetes and Metabolic Diseases (Prof T Yamada MD), University of Tokyo, Tokyo, Japan; Centre for Food and Nutrition Research, Institute of Medical Research and Plant Medicinal Studies, Yaounde, Cameroon (T B Ayuk PhD); Department of Health Studies, University of South Africa, Pretoria, South Africa (T B Ayuk PhD); Global Adolescent Health Group, Maternal and Child Health Program, Discipline of International Development, Burnet Institute, Melbourne, VIC, Australia (P S Azzopardi PhD); Department of Medical Mycology/Invasive Fungi Research Center (Prof H Badali PhD), Toxoplasmosis Research Center (Prof A Daryani PhD), Molecular and Cell Biology Research Center (Prof A Rafiei PhD), Department of Pediatrics (Prof M Rezaei MD), Department of Immunology (Prof A Rafiei PhD), Mazandaran University of Medical Sciences, Sari, Iran; Public Health Risk Sciences Division (A Badawi PhD), Health Promotion and Chronic Disease Prevention Branch (J J Lang PhD), Applied Research Division (H M Orpana PhD), Public Health Agency of Canada, Toronto, ON, Canada; Department of Hypertension, Medical University of Lodz, Lodz, Poland (Prof M Banach PhD); Polish Mother's Memorial Hospital Research Institute (PMMHRI), Lodz, Poland (Prof M Banach PhD); School of Psychology, University of Auckland, Auckland, New Zealand (Prof S L Barker-Collo PhD); Department of Industrial Engineering, Pontificia Universidad Javeriana, Bogota, Colombia (Prof L H Barrero DSc); University of Aden, Aden, Yemen (Prof H Basaleem PhD); Department of Public Health, Wollo University, Dessie, Ethiopia (E Baye MPH); Department of Psychiatry, Charles R. Drew University of Medicine and Science, Los Angeles, CA, USA (Prof S Bazargan-Hejazi BEP); Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA, USA (Prof S Bazargan-Hejazi BEP); Department of Community Medicine, Gandhi Medical College Bhopal, Bhopal, India (Prof N Bedi MD); Jazan University, Saudi Arabia (Prof N Bedi MD); Department of Neurology, University Hospital of Dijon, Dijon, France (Prof Y Béjot PhD); Dijon Stroke Registry, Faculty of Health Sciences, University of Burgundy, Dijon, France (Prof Y Béjot PhD); School of Public Health (A B Belachew MSc, G B Gebregers MPH), Institute of Biomedical Sciences, Department of Anatomy and Embryology, College of Health Sciences (T B Hagos MS), Biomedical Sciences Division, School of Medicine (Prof G B Hailu MSc, D T Mengistu MSc), Nutrition and Dietetics (A Kahsay MPH), Clinical Pharmacy Unit (T D D Kassa MSc, Y L Nirayo MSc, K G Weldegewergs MSc), School of Pharmacy (E Yimer MSc), Mekelle University, Mekelle, Ethiopia (Prof K G Meles MPH); Dr. Tewelde Legesse Health Sciences College, Mekelle, Ethiopia (S A Belay MPH); Department of Internal Medicine (I M Bensenor PhD, Prof P A Lotufo DrPH, Prof I S Santos PhD), Center for Clinical and Epidemiological Research (A C Goulart PhD), Department of Psychiatry (Y Wang PhD), University of São Paulo, São Paulo, Brazil; Dental Institute (E Bernabe PhD), Faculty of Life Sciences and Medicine (Prof P I Dargan MB), King's College London, London, UK; Hubert Department of Global Health, Emory University, Atlanta, GA, USA (R S Bernstein MD); Department of Global Health, University of South Florida, Tampa, Florida, USA (R S Bernstein MD); School of Public Health and Medicine, University of Newcastle, Newcastle, NSW, Australia (A S Beyene MPH, M Khan MSc); Air Pollution Research Center (B Heibati PhD), Community Medicine Department (Prof A Tehrani-Banihashemi PhD), Preventive Medicine and Public Health Research Center (Prof A Tehrani-Banihashemi PhD), Iran University of Medical Sciences, Tehran, Iran (T Beyranvand PhD); Research (Prof P K Maulik PhD), The George Institute for Global Health, New Delhi, India (S Bhaumik MBBS); Center of Excellence in Women and Child Health, Aga Khan University, Karachi, Pakistan (Prof Z A Bhutta PhD); Social Determinants of Health Research Center (A Bijani PhD), Health Research Institute (Prof R Ghadimi PhD, A Bijani PhD, A Rostami PhD), Department of Clinical Biochemistry (Prof H Parsian PhD), Infectious Diseases and Tropical Medicine Research Center (A Rostami PhD), Student Research Committee (M Zamani MD), Babol University of Medical Sciences, Babol, Iran (Prof M Faramarzi PhD); Woldia University, Woldia, Ethiopia (N Bililign BHLthSci); Department of Medical and Surgical Sciences (A Farioli PhD, Prof F S Violante MD), University of Bologna, Bologna, Italy (S M Birlik MBA); GBS/CIDP Foundation International, Conshohocken, PA, USA (S M Birlik MBA); The UCL Centre for Global Health Economics (C Birungi MSc), Department of Health Informatics (S Chung PhD), Department of Epidemiology and Public Health (Prof M Kivimäki PhD), University College London, London, UK; Fast-Track Implementation Department, United Nations Programme on HIV/AIDS (UNAIDS), Gaborone, Botswana (C Birungi MSc); Public Health, St Paul's Hospital Millennium Medical College, Addis Ababa, Ethiopia (H Bizuneh MPH); Department of Global Public Health and Primary Care, University of Bergen, Bergen, Norway (Prof T Bjørge PhD); Department of Research (Prof E Weiderpass PhD), Cancer Registry of Norway, Oslo, Norway (Prof T Bjørge PhD); Department of Epidemiology and Psychosocial Research, National Institute of Psychiatry Ramón de la Fuente Muñiz, Mexico City, Mexico (Prof G Borges DSc, R A Gutiérrez PhD); Department of Oncology (C Bosetti PhD), Department of Environmental Health Science (S Gallus DSc), Mario Negri Institute for Pharmacological Research (IRCCS), Milan, Italy; Transport and Road Safety (TARS) Research (S Boufous PhD), National Drug and Alcohol Research Centre (B Calabria PhD, Prof L Degenhardt PhD), School of Medicine (Prof P K Maulik PhD), School of Public Health and Community Medicine (Prof F Sitas PhD), University of New South Wales, Sydney, NSW, Australia; University of Genoa, Italy (N L Bragazzi PhD); Division of Clinical Epidemiology and Aging Research, German Cancer Research Center, Heidelberg, Germany (Prof H Brenner MD, B Schöttker PhD); School of Population and Public Health (Z A Butt PhD, F Pourmalek PhD, Prof N Sarrafzadegan MD), University of British Columbia, Vancouver, BC, Canada (C C Gotay PhD); Al Shifa School of Public Health, Al Shifa Trust Eye Hospital, Rawalpindi, Pakistan (Z A Butt PhD); School of Medicine, University of the Valley of Cuernavaca, Cuernavaca, Mexico (J Campuzano Rincon PhD); Institute for Cancer Research, Prevention and Clinical Network (ISPRO), Florence, Italy (G Carreras PhD); Applied Molecular Biosciences Unit (Prof F Carvalho PhD), Institute of Biomedical Engineering (INEB), (J Das Neves PhD), Department of Chemistry (Prof E Fernandes PhD), Institute for Research and Innovation in Health (i3s) (J Das Neves PhD), University of Porto, Porto, Portugal; Colombian National Health Observatory, National Institute of Health, Bogota, Colombia (C A Castañeda-Orjuela MSc); Epidemiology and Public Health Evaluation Group, National University of Colombia, Bogota, Colombia (C A Castañeda-Orjuela MSc); Costa Rican Department of Social Security, San Jose, Costa Rica (Prof J Castillo Rivas MSc); School of

- Dentistry, University of Costa Rica, San Pedro, Costa Rica (Prof J Castillo Rivas MSc); Department of Health Planning and Economics, National School of Public Health, Institute of Health Carlos III, Madrid, Spain (F Catalá-López PhD); College of Medicine, National Taiwan University, Taipei, Taiwan (Prof J Chang PhD); Department of Development Studies (Prof A Chattopadhyay PhD), Department of Public Health & Mortality Studies (Prof U Ram PhD), International Institute for Population Sciences, Mumbai, India (P Kumar PhD, A Patle MPH); Tata Memorial Hospital, Mumbai, India (Prof P Chaturvedi MD); Department of Public Health and Primary Care, University of Cambridge, UK (Prof R Chowdhury PhD); Department of Pulmonary Medicine (Prof D J Christopher FRCP), Department of Community Health (Prof A M Oommen MD), Department of Endocrinology (Prof N Thomas PhD), Christian Medical College and Hospital (CMC), Vellore, India; Health Data Research UK, London, UK (S Chung PhD); School of Medicine (L G Ciobanu PhD), University of Adelaide, Adelaide, SA, Australia (A T Olagunju MD); Department of Nutrition (Prof R M Claro PhD), Department of Maternal and Child Nursing and Public Health (Prof M S Felisbino-Mendes PhD, Prof D C Malta PhD), School of Nursing (I E Machado PhD), Federal University of Minas Gerais, Belo Horizonte, Brazil; Department of Medicine and Surgery, University of Milan-Bicocca, Monza, Italy (S Conti PhD, A Lafranchi MD, F Madotto PhD); Postgraduate Program in Epidemiology, Federal University of Rio Grande do Sul, Porto Alegre, Brazil (E Cousin MSc, B B Duncan MD, Prof M I Schmidt PhD); Department of Family Medicine and Public Health, University of California San Diego, La Jolla, CA, USA (Prof M H Criqui MD); Clinical Toxicology Service, Guy's and St. Thomas' NHS Foundation Trust, London, UK (Prof P I Dargan MB); Kazakh National Medical University, Almaty, Kazakhstan (Prof K Davletov PhD); Monash Centre for Health Research and Implementation (Prof B de Courten PhD), School of Public Health and Preventive Medicine (Prof Y Guo PhD), Centre of Cardiovascular Research and Education in Therapeutics (R Ofori-Asenso MSc), Monash University, Melbourne, VIC, Australia; Monash Health, Melbourne, VIC, Australia (Prof B de Courten PhD); Institute of Public Health (Prof J De Neve MD, B Moazen MSc, S Mohammed PhD), Department of Ophthalmology, Medical Faculty Mannheim (Prof J B Jonas MD), Heidelberg University, Heidelberg, Germany; Department of Clinical Pharmacy, Aksum University, Aksum, Ethiopia (G T Demoz MSc); Department of Psychiatry, Icahn School of Medicine at Mount Sinai, New York, NY, USA (Prof D C Des Jarlais PhD); Disha Foundation, Gurgaon, India (S Dey PhD); Indian Council of Medical Research, New Delhi, India (R S Dhalwal MD); Department of Community Medicine, University of Peradeniya, Peradeniya, Sri Lanka (S D Dharmaratne MD); Health Research Section, Nepal Health Research Council, Kathmandu, Nepal (M Dhimal PhD); Department of Population and Health, University of Cape Coast, Cape Coast, Ghana (D T Doku PhD); School of Health Sciences, University of Tampere, Tampere, Finland (D T Doku PhD, S Neupane PhD); School of Health and Biomedical Sciences, Royal Melbourne Institute of Technology (RMIT) University, Bundoora, VIC, Australia (Prof K E Doyle PhD); United Nations World Food Programme, New Delhi, India (M Dubey PhD); Faculty of Medicine, University of Belgrade, Serbia (E Dubljanin PhD); Department of Clinical Pathology, Mansoura University, Mansoura, Egypt (Prof M El Sayed Zaki PhD); Laboratory for Socio-Economic Issues of Human Development and Quality of Life, Russian Academy of Sciences, Moscow, Russia (Prof S P Ermakov DSc); Department of Medical Statistics and Documentation (Prof S P Ermakov DSc), Federal Research Institute for Health Organization and Informatics of the Ministry of Health (FRIHOI), Moscow, Russia (S K Vladimirov PhD); Policy and Epidemiology Group (D F Santomauro PhD), Queensland Centre for Mental Health Research, Brisbane, QLD, Australia (H E Erskine PhD, A J Ferrari PhD); Department of Psychology, Federal University of Sergipe, Sao Cristovao, Brazil (Prof A Faro PhD); National Institute for Stroke and Applied Neurosciences, Auckland University of Technology, Auckland, New Zealand (Prof V L Feigin PhD); Federal University of São Paulo, São Paulo, Brazil (C P Ferri PhD); Heller School for Social Policy and Management, Brandeis University, Waltham, MA, USA (D O Fijabi MS); School of Public Health, University of Memphis, Memphis, Tennessee, USA (D O Fijabi MS); Kaiser Permanente, Fontana, CA, USA (I Filip MD); Department of Health Sciences (I Filip MD), A.T. Still University, Mesa, Arizona, USA (A Radfar MD); Department of Epidemiology and Health Monitoring, Robert Koch Institute, Berlin, Germany (J D Finger PhD); Department of Public Health Medicine, Bielefeld University, Bielefeld, Germany (F Fischer PhD); College of Public Health, Medical and Veterinary Science, James Cook University, Townsville, QLD, Australia (Prof R C Franklin PhD); Neurosurgery Department, Sidi Mohamed Ben Abdellah University, Fez, Morocco (F G Gankpe MD); Division of Human Nutrition and Health, Wageningen University & Research, Wageningen, Netherlands (Prof J M Geleijnse PhD); Department of Health Care Policy & Management, University of Tsukuba, Tsukuba, Japan (M Ghimire MA); Unit of Academic Primary Care (Prof P S Gill DM), Division of Health Sciences (Prof O A Uthman PhD), University of Warwick, Coventry, UK; Department of Family and Community Medicine, University of Hail, Hail, Saudi Arabia (Prof I A Ginawi MD); College of Nursing and Health Sciences, University of Massachusetts Boston, Boston, MA, USA (Prof P N Gona PhD); Department of Biostatistics and Epidemiology, University of Oklahoma, Oklahoma City, OK, USA (S V Gopalani MPH); Department of Health and Social Affairs, Government of the Federated States of Micronesia, Palikir, Federated States of Micronesia (S V Gopalani MPH); Department of Primary Care and Public Health (F Greaves PhD, Prof A Majeed MD, Prof S Rawaf PhD), WHO Collaborating Centre for Public Health Education & Training (D L Rawaf MD), School of Public Health (Prof S Saxena MD), Imperial College London, London, UK; Health Improvement Directorate (F Greaves PhD), Public Health England, London, UK (Prof S Rawaf PhD); Integrated Tumor Registry, University-Hospital Policlinic Vittorio Emanuele, Catania, Italy (G Grosso PhD); Commissioner of Public Health, West Virginia Bureau for Public Health, Charleston, WV, USA (Prof R Gupta MD); Department of Health Policy, Management & Leadership, West Virginia University, Morgantown, WV, USA (Prof R Gupta MD); Rajasthan University of Health Sciences, Jaipur, India (Prof R Gupta MD); Department of Preventive Cardiology, Eternal Heart Care Centre & Research Institute, Jaipur, India (Prof R Gupta MD); Department of Anthropology, University of Delhi, Delhi, India (V Gupta PhD); Indian Institute of Public Health, Public Health Foundation of India, Hyderabad, Hyderabad, India (Prof M GVS MD); Department of Radiology, Johns Hopkins University, Baltimore, MD, USA (N Hafezi-Nejad MD); Department of Family and Community Medicine, Arabian Gulf University, Manama, Bahrain (Prof R R Hamadeh DPhil); School of Health and Environmental Studies, Hamdan Bin Mohammed Smart University, Dubai, United Arab Emirates (Prof S Hamidi DrPH); Neurology Department, Sir Charles Gairdner Hospital, Perth, WA, Australia (Prof G J Hankey MD); Department of Vital and Health Statistics (H L Harb MPH), Department of Disease, Epidemics and Pandemics Control (J Nansseu MD), Ministry of Public Health, Beirut, Lebanon; Cardiology Department (Prof S Harikrishnan MD), Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India (G Mini PhD); Research and Development Unit, Parc Sanitari Sant Joan de Déu (CIBERSAM), Sant Boi de Llobregat, Spain (Prof J M Haro MD, A Koyanagi MD, S Tyrovolas PhD); Department of Medicine (Prof J M Haro MD), University of Barcelona, Barcelona, Spain (S Tyrovolas PhD); Public Health Department (H Y Hassen MPH), Pharmacy Department (A T Kefale MSc), Mizan-Tepi University, Mizan Teferi, Ethiopia (Prof A Henok MPH); Unit of Epidemiology and Social Medicine, University Hospital Antwerp, Wilrijk, Belgium (H Y Hassen MPH); Karolinska University Hospital, Stockholm, Sweden (R Havmoeller PhD); Subdirector of Regulations, Guidelines and Technical Procedures, Secretary of Health, National Commission Against Addiction, Mexico City, Mexico (Prof N F Hernández-Llanes MPH); Department of Statistics and Econometrics (Prof C Herteliu PhD, Prof A Mirica PhD, A Pana MD), Bucharest University of Economic Studies, Bucharest, Romania (B V Ileanu PhD); Department of Reproductive Health, Hawassa University, Hawassa Ethiopia (D T T Hibstu MPH); Transdisciplinary Centre for Qualitative Methods, Manipal University, Manipal, India (P Hoogar PhD); Department of Pulmonology, Yokohama City University, Yokohama, Japan (N Horita MD); National Human Genome

Research Institute (NHGRI), National Institutes of Health, Bethesda, MD, USA (N Horita MD); Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY, USA (Prof H Hosgood PhD); Department of Internal Medicine, Emergency Hospital of Bucharest, Bucharest, Romania (Prof M Hostiu PhD); Department of Epidemiology and Health Statistics, Xiangya School of Public Health, Central South University, Changsha, China (Prof G Hu PhD); Department of Psychiatry, Cambridge Health Alliance, Cambridge, MA, USA (H Huang MD); Institute of Community and Public Health, Birzeit University, Birzeit, Palestine (Prof A Hussein PhD); Qatar University, Doha, Qatar (Prof A Hussein PhD); Infectious Diseases Department, Bashkir State Medical University, Ufa, Russia (B Idrisov MD); Department of Public Health and Community Medicine, University of Liberia, Monrovia, Liberia (O S Ilesanmi PhD); Research Institute for Endocrine Sciences, Shahid Beheshti University of Medical Sciences, Tehran, Iran (S N Irvani MD); Institute for Physical Activity and Nutrition (S Islam PhD), National Centre for Farmer Health, School of Medicine (M Rahman PhD), Deakin University, Warrnambool, VIC, Australia; Department of Community Health and Psychiatry, University of the West Indies, Mona, Jamaica (Prof M D Jackson PhD); Faculty of Medical Sciences, University of Kragujevac, Kragujevac, Serbia (Prof M Jakovljevic PhD); Faculty of Graduate Studies (A U Jayatilake PhD), Postgraduate Institute of Medicine (A U Jayatilake PhD), University of Colombo, Colombo, Sri Lanka; Department of Community Medicine, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India (R P Jha MSc); Capital Medical University, Beijing Institute of Ophthalmology, Beijing, China (Prof J B Jonas MD); Institution of Health and Nutrition Sciences, Czestochowa University of Technology, Czestochowa, Poland (Prof J J Jozwiak PhD); Faculty of Medicine and Health Sciences, University of Opole, Opole, Poland (Prof J J Jozwiak PhD); School of Public Health, University College Cork, Cork, Ireland (Z Kabir PhD); Department of Health Policy, Personal Social Services Research Unit, London School of Economics and Political Science, London, UK (R Kadel MPH); A.C.S. Medical College and Hospital, New Delhi, India (Prof U Kapil MD, Prof Z Zaidi DrPH); MRC/CSO Social & Public Health Sciences Unit, University of Glasgow, Glasgow, UK (S V Katikireddi PhD); Midwifery Program, Salale University, Fiche, Ethiopia (S Kebede MSc); Odel Campus, University of Nairobi, Nairobi, Kenya (Prof P N Keiyoro PhD); Non-Communicable Diseases Research Unit (Prof A P Kengne PhD), Alcohol, Tobacco & Other Drug Use Research Unit (Prof C D H Parry PhD), Medical Research Council South Africa, Cape Town, South Africa; Department of Medicine (Prof A P Kengne PhD, J Noubiap MD), Groote Schuur Hospital (G A Mensah MD), Department of Psychiatry and Mental Health (Prof D J Stein MD), University of Cape Town, Cape Town, South Africa; Department of Public Health and Community Medicine, Jordan University of Science and Technology, Alramtha, Jordan (Prof Y Khader PhD); Department of Public Health (Prof M A Khafae PhD), Health Research Institute, Thalassemia and Hemoglobinopathy Research Center (Prof F Rahim PhD), Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran; Department of Population Sciences, Jatiya Kabi Kazi Nazrul Islam University, Mymensingh, Bangladesh (M Khan MSc); Institute of Health Policy and Management, SNU Medical Research Center (Prof Y Khang MD), Department of Health Policy and Management (Prof Y Khang MD), Seoul National University, Seoul, South Korea; Department of Medical Parasitology, Cairo University, Cairo, Egypt (M M Khater MD); Department of Nutrition and Health Science, Ball State University, Muncie, IN, USA (Prof J Khubchandani PhD); Korea Health Industry Development Institute, Cheongju-Si, South Korea (C Kim PhD); Department of Health Sciences, Northeastern University, Boston, MA, USA (Prof D Kim DrPH); School of Medicine, Xiamen University Malaysia, Sepang, Malaysia (Prof Y Kim PhD); Department of Nutrition, Simmons College, Boston, MA, USA (R W Kimokoti MD); Department of Health Management and Health Economics (Prof A Kisa PhD), Norwegian Center for Addiction Research (SERAF), (S P Neupane PhD), University of Oslo, Oslo, Norway; Department of Global Community Health and Behavioral Sciences, Tulane University, New Orleans, LA, USA (Prof A Kisa PhD); Department of Public Health

(Prof M Kivimäki PhD, T Lallukka PhD), University of Helsinki, Helsinki, Finland (T J Meretoja MD); Department of Public Health, Erasmus University Medical Center Rotterdam, The Netherlands, Erasmus University Medical Center, Netherlands (S Kochhar MD); International Institute of Health Management Research, New Delhi, India (A Patle MPH); Independent Consultant, Jakarta, Indonesia (S Kosen MD); Department of Internal and Pulmonary Medicine, Sheri Kashmir Institute of Medical Sciences, Srinagar, India (Prof P A Koul MD); Department of Anthropology, Panjab University, Chandigarh, India (Prof K Krishan PhD); Department of Social and Preventive Medicine (Prof B Kuate Defo PhD), Department of Demography (Prof B Kuate Defo PhD), University of Montreal, Montreal, QC, Canada; Faculty of Medicine, Department of Public Health, Yuksek Ihtisas University, Ankara, Turkey (Prof B Kucuk Bicer BEP); Faculty of Medicine, Department of Public Health, Hacettepe University, Ankara, Turkey (Prof B Kucuk Bicer BEP); Arkansas State University, USA (V S Kulkarni PhD); School of Population and Global Health (Prof A D Lopez PhD), Department of Paediatrics (Prof G C Patton MD), Australian Institute of Muscular Skeletal Sciences, Department of Medicine and Neurology (Prof T Wijeratne MD), University of Melbourne, Melbourne, VIC, Australia; Department of Community Medicine (M S Nayak MD), Rajiv Gandhi University of Health Sciences, Bangalore, India (A Lakshmana Balaji MPH); Population and Work Ability Program (T Lallukka PhD), Finnish Institute of Occupational Health, Helsinki, Finland (R Shiri PhD); Institute of Health Policy and Development Studies, National Institutes of Health, Manila, Philippines (Prof H Lam PhD); Department of Community and Family Medicine (Prof F H Lami PhD), Academy of Medical Science, Baghdad, Iraq; Division of Cancer Epidemiology & Genetics, National Cancer Institute, Rockville, MD, USA (Q Lan PhD); Belo Horizonte City Hall, Municipal Health Department of Belo Horizonte, Belo Horizonte, Brazil (Prof S Lansky PhD); Department of Medical Sciences, Uppsala University, Uppsala, Sweden (Prof A O Larsson PhD); Department of Clinical Chemistry and Pharmacology, Akademiska Sjukhuset, Uppsala, Sweden (Prof A O Larsson PhD); Managerial Epidemiology Research Center, Department of Public Health, School of Nursing and Midwifery (S Safiri PhD), Department of Public Health, School of Public Health, (A Latifi PhD), Maragheh University of Medical Sciences, Maragheh, Iran; College of Optometry, Nova Southeastern University, Fort Lauderdale, FL, USA (J L Leasher OD); School of Nursing, Hong Kong Polytechnic University, Hong Kong, China (P H Lee PhD); Asbestos Diseases Research Institute (J Leigh MD), School of Public Health (Prof F Sitas PhD), University of Sydney, Sydney, NSW, Australia; Scihost, Södertörn University, Huddinge, Sweden (Prof M Leinsalu PhD); Department of Epidemiology and Biostatistics, National Institute for Health Development, Tallinn, Estonia (Prof M Leinsalu PhD); CERIMP, Local Health Unit Tuscany Centre, Florence, Italy (M Levi PhD); Department of Health Sciences, University of Florence, Florence, Italy (M Levi PhD); Department of Clinical and Epidemiological Research, Shenzhen Institute of Cardiovascular Disease, Shenzhen, China (Prof Y Li PhD); Department of Medicine, University of Malaya, Kuala Lumpur, Malaysia (L Lim MRCP); Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Shatin, China (L Lim MRCP); School of Public Health, University of Haifa, Haifa, Israel (Prof S Linn DrPH); Centre for Chronic Disease Control, China (Prof S Liu PhD); National Institute of Energy Efficiency and Renewable Energies (INER), Quito, Ecuador (A Lobato-Cordero MSc); Damietta University, Damietta, Egypt (H Magdy Abd El Razek MD); Ophthalmology Department, Aswan Faculty of Medicine, Aswan, Egypt (M Magdy Abd El Razek MB); Department of Public Health, Trnava University, Trnava, Slovakia (Prof M Majdan PhD); Non-Communicable Diseases Research Center, Shiraz University of Medical Sciences, Shiraz, Iran (Prof R Malekzadeh MD, S G Sepanlou MD); Department of Population Studies, University of Zambia, Lusaka, Zambia (C Mapoma PhD); Psychiatry Department, Doctor Peset University Hospital, Valencia, Spain (J Martinez-Raga MD); Department of Medicine (J Martinez-Raga MD, Prof R Tabarés-Seisdedos PhD), University of Valencia, Valencia, Spain; Department of Biology and Biological Engineering, Chalmers University of Technology, Gothenburg, Sweden (M Mazidi PhD); Department of

- Health Services Research & Policy (Prof M McKee DSc), Department of Non-communicable Disease Epidemiology (Prof D Prabhakaran DM), London School of Hygiene & Tropical Medicine, London, UK; Department of Internal Medicine, Sevenhills Hospital, Mumbai, India (V Mehta MD); Institute for Agricultural and Nutritional Sciences (T Meier PhD), Institute of Epidemiology, Biostatistics and Informatics (I Shiu PhD), Martin Luther University Halle-Wittenberg, Halle, Germany; Innovation Office Nutricard, Competence Cluster for Nutrition and Cardiovascular Health (NUTRICARD), Halle, Germany (T Meier PhD); College of Health Sciences, Debre Tabor University, Debre Tabor, Ethiopia (A Melese MSc); Department of Public Health, University of West Florida, Pensacola, FL, USA (Prof P T N Memiah DrPH); Peru Country Office of the UNFPA, United Nations Population Fund (UNFPA), Lima, Peru (W Mendoza MD); Center for Translation Research and Implementation Science, National Heart, Lung, and Blood Institute, Bethesda, MD, USA (G A Mensah MD); Comprehensive Cancer Center, Breast Surgery Unit, Helsinki University Hospital, Helsinki, Finland (T J Meretoja MD); Ethiopian Academy of Medical Science, Mekelle, Ethiopia (Prof H B Mezgebe MSc); Department of Hypertension & Internal Medicine (Prof T Miazgowski MD), Zdroje Hospital (J Widecka PhD), Pomeranian Medical University, Szczecin, Poland; Pacific Institute for Research & Evaluation, Calverton, MD, USA (T R Miller PhD); School of Public Health, Curtin University, Perth, WA, Australia (T R Miller PhD); Department of Public Health, Amrita Institute of Medical Sciences, Kochi, India (G Mini PhD); President's Office, National Institute of Statistics Romania, Bucharest, Romania (Prof A Mirica PhD); Kyrgyz State Medical Academy, Bishkek, Kyrgyzstan (Prof E M Mirakshimov MD); Department of Atherosclerosis and Coronary Heart Disease, National Center of Cardiology and Internal Disease, Bishkek, Kyrgyzstan (Prof E M Mirakshimov MD); Department of Health and Social Work, Institute of Addiction Research (ISFF), Frankfurt University of Applied Sciences, Frankfurt Am Main, Germany (B Moazen MSc); Department of Biology, Salahaddin University, Erbil, Iraq (K A Mohammad PhD); ISHIK University, Erbil, Iraq (K A Mohammad PhD); Nutrition and Cohort Studies Department, Isfahan Cardiovascular Research Institute (N Mohammadifard PhD), Cardiovascular Research Institute (Prof N Sarrafzadegan MD), Isfahan University of Medical Sciences, Isfahan, Iran; Health Systems and Policy Research Unit (S Mohammed PhD), Department of Community Medicine (M B Sufiyan MD), Ahmadu Bello University, Zaria, Nigeria; Clinical Epidemiology and Public Health Research Unit, Institute for Maternal and Child Health, IRCCS Burlo Garofolo, Trieste, Italy (L Monasta DSc, L Ronfani PhD); Lancaster University, UK (P Moraga PhD); International Laboratory for Air Quality and Health, Science and Engineering Faculty, Queensland University of Technology, Brisbane, QLD, Australia (Prof L Morawska PhD); St. Paul's Hospital Millennium Medical College, Addis Ababa, Ethiopia (M T Jalu MPH); Department of Endocrinology & Metabolism, Institute of Postgraduate Medical Education and Research, Kolkata, Kolkata, India (Prof S Mukhopadhyay MD); School of Medical Sciences, University Sains Malaysia, Kubang Kerian, Malaysia (Prof K Musa PhD); Initiative for Non Communicable Diseases, International Centre for Diarrhoeal Disease Research, Bangladesh, Dhaka, Bangladesh (A Naheed PhD); Department of Epidemiology, University of Alabama at Birmingham, Birmingham, AL, USA (G Naik MPH); Epidemiology Department (Prof F Najafi PhD), Research Center for Environmental Determinants of Health (M Soofi PhD), Kermanshah University of Medical Sciences, Kermanshah, Iran; Suraj Eye Institute, Nagpur, India (V Nangia MD); Department of Public Health, Faculty of Medicine and Biomedical Sciences, University of Yaoundé, Yaoundé, Cameroon (J Nansseu MD); Department of Epidemiology and Public Health, University Sidi Mohammed Ben Abdellah, Fez, Morocco (Prof C Nejari PhD); International School of Public Health, Mohammed VI University of Health Science, Casablanca, Morocco (Prof C Nejari PhD); Norwegian National Advisory Unit on Concurrent Substance Abuse and Mental Health Disorders, Innlandet Hospital Trust, Brumunddal, Norway (S P Neupane PhD); Department of Biological Sciences, University of Embu, Embu, Kenya (J W Ngunjiri DrPH); Institute for Global Health Innovations, Duy Tan University, Hanoi, Vietnam (C T Nguyen MPH, L H Nguyen MPH, T H Nguyen BMedSc); Public Health Department, Semarang State University, Kota Semarang, Indonesia (D N A Ningrum MPH); Graduate Institute of Biomedical Informatics, Taipei Medical University, Taipei City, Taiwan (D N A Ningrum MPH); Research Unit, Health Policy Consult, Accra, Ghana (R Ofori-Asenso MSc); School of Social Sciences and Psychology Department (Prof A M N Renzaho PhD), Western Sydney University, Penrith, NSW, Australia (F A Ogbo PhD); Dept. of Preventive Medicine, School of Medicine, Kyung Hee University, Dongdaemun-Gu, South Korea (Prof I Oh PhD); HIV/AIDS, STIs & TB (HAST) Programme, Human Sciences Research Council (HSRC), Durban, South Africa (O Oladimeji MD); School of Public Health, Faculty of Health Sciences, University of Namibia, Osakhaty, Namibia (O Oladimeji MD); Department of Psychiatry, University of Lagos, Lagos, Nigeria (A T Olagunju MD); Autonomous University of Chile, Chile (Prof P R Olivares PhD); Executive Director (B O Olusanya PhD), Centre for Healthy Start Initiative, Ikoyi, Nigeria (J O Olusanya MBA); Graduate School of Public Health, San Diego State University, San Diego, CA, USA (Prof E Oren PhD); School of Psychology, University of Ottawa, Ottawa, ON, Canada (H M Orpana PhD); Center for Vaccine Development, University of Maryland, Baltimore, MD, USA (Prof J R Ortiz MD); Global Health Nursing, St. Luke's International University, Chuo-Ku, Japan (Prof E Ota PhD); College of Medicine, University of Ibadan, Ibadan, Nigeria (Prof M O Owolabi DrM); Agricultural Economics Group (Prof A S Oyekale PhD), Hypertension in Africa Research Team (HART), (Prof A E Schutte PhD), North-West University, Mafikeng, South Africa; Department of TB and Respiratory Medicine, Jagadguru Sri Shivarathreeswara University, Mysore, India (Prof M P A DNB); Center for Health Outcomes & Evaluation, Bucharest, Romania (A Pana MD); Department of Medical Humanities and Social Medicine, Kosin University, Busan, South Korea (Prof E Park PhD); Population Health Group, Murdoch Children's Research Institute, Melbourne, VIC, Australia (Prof G C Patton MD); Health, Nutrition and HIV AIDS Program, Save the Children, Kathmandu, Nepal (D Paudel PhD); Center for International Health, Ludwig Maximilians University, Munich, Germany (D Paudel PhD); Institute of Medicine, University of Gothenburg, Gothenburg, Sweden (Prof M Petzold PhD); School of Public Health, University of Witwatersrand, Johannesburg, South Africa (Prof M Petzold PhD); Shanghai Mental Health Center, Shanghai Jiao Tong University, Shanghai, China (Prof M R Phillips MD); Basic Medical Sciences Department, Durban University of Technology, Durban, South Africa (Prof J D Pillay PhD); Department of Economics & Business (Prof M J Postma PhD), University Medical Center Groningen (Prof J F M Van Boven PhD, Prof M J Postma PhD), University of Groningen, Groningen, Netherlands; Non-Communicable Diseases Research Center, Alborz University of Medical Sciences, Karaj, Iran (M Qorbani PhD); Medichem, Spain (A Radfar MD); Contech School of Public Health, Lahore, Pakistan (A Rafay MS, Prof S M Rana PhD); Research and Evaluation Division, BRAC, Dhaka, Bangladesh (M Rahman PhD); Austin Clinical School of Nursing (M Rahman PhD), Centre for Alcohol Policy Research (Prof R Room PhD), Department of Psychology and Counselling (Prof T Wijeratne MD), La Trobe University, Heidelberg, VIC, Australia; Society for Health and Demographic Surveillance, Suri, India (R Rai MPH); Department of Economics, University of Göttingen, Göttingen, Germany (R Rai MPH); Medical University Innsbruck, Austria (S Rajsic MD); Department of Nephrology, Nizam's Institute of Medical Sciences, Hyderabad, India (Prof S Raju MD); Public Health Department, University of Health Sciences, Lahore, Pakistan (Prof S M Rana PhD); Institute for Poverty Alleviation and International Development (C L Ranabhat PhD), Yonsei University, Seoul, Korea; University College London Hospitals, UK (D L Rawaf MD); Department of Preventive Medicine and Occupational Medicine, Loma Linda University Medical Center, Loma Linda, CA, USA (C Reis MD); Department of Clinical Research, University of Uberlândia, Uberlândia, Brazil (L Roeber PhD); Centre for Social Research on Alcohol and Drugs, Department of Public Health Science, Stockholm University, Stockholm, Sweden (Prof R Room PhD); Golestan Research Center of Gastroenterology and Hepatology, Golestan University of Medical Sciences, Gorgan, Iran (G Roshandel PhD); Department of Cardiology, All India Institute of Medical Sciences (AIIMS), New Delhi, India (Prof A Roy MD); National Institute for Research in Environmental

Health, Indian Council of Medical Research, Bhopal, India (Y D Sabde MD); College of Medicine, University of Sharjah, Sharjah, United Arab Emirates (Prof B Saddik PhD); Punjab University College of Pharmacy, Pakistan (Z Saleem PharmD); Center for Health Policy & Center for Primary Care and Outcomes Research, Stanford University, Stanford, CA, USA (Prof J A Salomon PhD); Clinical Research Division, Chest Research Foundation, Pune, India (Prof S S Salvi MD); Department of Surgery, Marshall University, Huntington, WV, USA (Prof J Sanabria MD); Department of Nutrition and Preventive Medicine, Case Western Reserve University, Cleveland, OH, USA (Prof J Sanabria MD); Nephrology Group, LIS-Fundación Jimenez Diaz, Madrid, Spain (M Sanchez-Niño PhD); Faculty of Medicine, Institute of Social Medicine, Centre School of Public Health and Health Management, University of Belgrade, Belgrade, Serbia (Prof M M M Santric Milicevic PhD); Health Economics and Financing Research Group, International Centre for Diarrhoeal Disease Research, Bangladesh, Dhaka, Bangladesh (A R Sarker MSc); Department of Health and Society, Faculty of Medicine, University of Applied and Environmental Sciences, Bogotá, Colombia (Prof R Sarmiento-Suárez MPH); Department of Public Health Medicine, Howard College Campus, University of Kwazulu-Natal, Durban, South Africa (Prof B Sartorius PhD); UGC Centre of Advanced Study in Psychology, Utkal University, Bhubaneswar, India (Prof M Satpathy PhD); Udyam-Global Association for Sustainable Development, Bhubaneswar, India (Prof M Satpathy PhD); Department of Public Health Sciences, University of North Carolina at Charlotte, Charlotte, NC, USA (M Sawhney PhD); Market Access, Bayer, Istanbul, Turkey (M Saylan MD); Swiss Research Institute for Public Health and Addiction, University of Zürich, Zürich, Switzerland (M P Schaub PhD); Health Sciences Department, Federal University of Santa Catarina, Florianópolis, Brazil (Prof I J C Schneider PhD, Prof D A S Silva PhD); Department of Operative and Preventive Dentistry, Charity University Medical Center - Berlin, Berlin, Germany (Prof F Schwendicke MPH); Independent Consultant, Karachi, Pakistan (M A Shaikh MD); Department of Laboratory Sciences (Prof M Sharif PhD), Department of Basic Sciences (Prof M Sharif PhD), Islamic Azad University, Sari, Iran; Department of Pulmonary Medicine, Fudan University, Shanghai, China (J She MD); Usher Institute of Population Health Sciences and Informatics, University of Edinburgh, Edinburgh, UK (Prof A Sheikh MSc); The Research Institute at Nationwide Children's Hospital, Columbus, OH, USA (J Shen PhD); National Institute of Infectious Diseases, Tokyo, Japan (M Shigematsu PhD); Washington State University, Pullman, WA, USA (Prof K Shishani PhD); Symbiosis Institute of Health Sciences, Symbiosis International University, Pune, India (Prof S R Shukla PhD); Department of Psychology, Reykjavik University, Reykjavik, Iceland (Prof I D Sigfusdottir PhD); Department of Health and Behavior Studies, Columbia University, New York, New York, USA (Prof I D Sigfusdottir PhD); Portuguese Institute of Sport and Youth, Lisbon, Portugal (N T da Silva MPsyCh); Brasília University, Brasília, Brazil (Prof D A Silveira MSc); School of Preventive Oncology, Patna, India (D N Sinha PhD); Department of Epidemiology, Healis Sekhsaria Institute for Public Health, Mumbai, India (D N Sinha PhD); Pneumology Service, Research Institute of the University Hospital of the Princess (IISP), Madrid, Spain (Prof J B Soriano MD); Pneumology Service, Autonomous University of Madrid, Madrid, Spain (Prof J B Soriano MD); Division of Community Medicine, International Medical University, Kuala Lumpur, Malaysia (Prof C T Sreeramareddy MD); Institute and Outpatient Clinic for Occupational, Social and Environmental Medicine, University Hospital Munich, Munich, Germany (N Steckling DrPH); Department of Public Health, Health Services Research and Health Technology Assessment, University for Health Sciences, Medical Informatics and Technology, Hall I.T., Austria (N Steckling DrPH); Department of Criminology, Law and Society, University of California Irvine, Irvine, CA, USA (Prof B L Sykes PhD); Carlos III Health Institute, CIBERSAM, Madrid, Spain (Prof R Tabarés-Seisdedos PhD); Cancer Control Center, Osaka International Cancer Institute, Osaka, Japan (T Tabuchi MD); Department of Psychiatry and Behavioral Sciences, New York Medical College, Valhalla, NY, USA (M Tavakkoli MD); Institute of Public Health, Jagiellonian University Medical College, Krakow, Poland (R Topor-Madry PhD); Department of Ophthalmology, Medical School,

Aristotle University of Thessaloniki, Thessaloniki, Greece (Prof F Topouzis PhD); Department of Health Economics, Hanoi Medical University, Hanoi, Vietnam (Prof B X Tran PhD); Department of Neurology, University of Copenhagen, Copenhagen, Denmark (T C Truelsen PhD); Department of Vascular Medicine, University Heart Center of Hamburg, Hamburg, Germany (Prof N Tsimilparis PhD); Department of Internal Medicine, Federal Teaching Hospital, Abakaliki, Nigeria (K N Ukwaja MSc); Gomal Centre of Biochemistry and Biotechnology, Gomal University, Dera Ismail Khan, Pakistan (I Ullah PhD); Programmatic Management of Drug Resistant TB Unit, TB Culture Laboratory, Mufti Mehmood Memorial Teaching Hospital, Dera Ismail Khan, Pakistan (I Ullah PhD); Argentine Society of Medicine, Buenos Aires, Argentina (Prof P R Valdez MEd); Intensive Care Unit, Hospital Velez Sarsfield, Buenos Aires, Argentina (Prof P R Valdez MEd); UKK Institute, Tampere, Finland (Prof T J Vasankari MD); Raffles Neuroscience Centre, Raffles Hospital, Singapore, Singapore (Prof N Venketasubramanian FRCP); Department of Information and Internet Technologies, I.M. Sechenov First Moscow State Medical University, Moscow, Russia (S K Vladimirov PhD); Department of Health Care Administration and Economy, National Research University Higher School of Economics, Moscow, Russia (Prof V Vlassov MD); Center for Disease Burden, Norwegian Institute of Public Health, Bergen, Norway (Prof S Vollset DrPH); Department of Nursing, Debre Markos University, Debre Markos, Ethiopia (F W S Wagnew MSc); Foundation University Medical College, Foundation University, Rawalpindi, Pakistan (Prof Y Waheed PhD); Demographic Change and Ageing Research Area (A Werdecker PhD), Competence Center of Mortality-Follow-Up, German National Cohort (R Westerman DSc), Federal Institute for Population Research, Wiesbaden, Germany; Independent Consultant, Satufenberg, Germany (A Werdecker PhD); Information Services Division, NHS Scotland, Edinburgh, Scotland (G M A Wyper MSc); University of Strathclyde, Glasgow, Scotland (G M A Wyper MSc); School of Medicine, Nanjing University, Nanjing, China (Prof G Xu MD); Department of Preventive Medicine, University of Mississippi Medical Center, Jackson, MS, USA (Prof Y Yano MD); Division of Injury Prevention and Mental Health Improvement, Chinese Center for Disease Control and Prevention, Beijing, China (P Ye MPH); Centre for Suicide Research and Prevention, University of Hong Kong, Hong Kong, China (Prof P Yip PhD); University of South Australia, Adelaide, Australia (B D Yirsaw PhD); Department of Biostatistics, School of Public Health, Kyoto University, Kyoto, Japan (Prof N Yonemoto MPH); Department of Preventive Medicine, Korea University, Seoul, South Korea (Prof S Yoon PhD); College of Public Health, Ohio State University, Columbus, OH, USA (Prof M Yotebieng PhD); School of Public Health, University of Kinshasa, Kinshasa, Democratic Republic of the Congo (Prof M Yotebieng PhD); Department of Health Policy and Management, Jackson State University, Jackson, MS, USA (Prof M Z Younis DrPH); Tsinghua University, Beijing, China (Prof M Z Younis DrPH); Department of Cardiology, Mother Hospital, Thrissur, India (G Zachariah MD); University of Texas, Houston, TX, USA (X Zhang PhD).

Contributors

Max G Griswold, Nancy Fullman, and Emmanuela Gakidou prepared the manuscript. Max G Griswold, Nancy Fullman, and Stephanie R M Zimsen extracted data. Max G Griswold developed the models, methods, conducted all analysis and produced the results. Emmanuela Gakidou, Mohammad H Forouzanfar, and Christopher J L Murray conceived of the study. Emmanuela Gakidou provided overall guidance. Caitlin Hawley and Joseph S Salama managed the project. All other authors contributed to at least one of the following: provided data, reviewed results, developed modelling infrastructure, and/or reviewed and contributed to the final manuscript.

Declaration of interests

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Data sharing

Raw data underlying figures and relative risk curves have been made publicly available on Mendeley Data, a secure online repository for research data, as of September 19, 2018 (DOI:10.17632/5thy2mcwn7.1).

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